Food matters: Translating information on what people eat, say they eat, into advice for health

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DEDICATION

To the memory of Magdalena Gallegos and Georgino Lara

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DECLARATION OF PERSONAL INVOLVEMENT AND EXTENT OF COLLABORATION IN THE PRESENT THESIS

I declare that I have personally designed and conducted all the studies included in the present thesis. I have personally carried out all the measurements and collected and analysed all data for every single study in this thesis.

Some data contained in chapter 2 were gathered during my MSc course (supervised by Prof. Michael Lean) and included in thesis to obtain the degree of MSc. Data presented in chapter 2 have been expanded and re-analysed.

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Jose J. Lara Gallegos

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µmol	Micromol
♀	Women
♂	Men
24-hR	twenty-four hour recall
3-FEQ	Three Factor Eating Questionnaire
7-d	Seven-day
Alc	Alcohol
ANOVA	Analisys of Variance
ApoE	Apolipoprotein E
BMI	Body Mass Index
BMR	Basal Metabolic Rate
BMRest	Basal Metabolic Rate estimated (Through a formula or equation)
BODPOD	Body composition analysis through the Air Displacement Plethysmography method
CHD	Coronary Herat Disease
CHO	Carbohydrate
CRP	C-Reactive Protein
CVD	Cardio-Vascular Disease
DEPCAT	Deprivation Category
DH	Dietary History
DHA	Docosahexaenoic Acid

DLW	Double Labelled Water
DN	Dietary Nitrogen
DRVs	Dietary Reference Values
DTM	Dietary targets Monitor
EI	Energy Intake
EI:BMR	Energy Intake/basal metabolic Rate (ratio)
EPA	Eicosapentaenoic acid
FFQ	Food Frequency Questionnaire
GC/MC	Gas Chromatograpphy/ Mass Spectrometry
HDL	High Density Lipoprotein (Cholesterol)
HOMA	Homeostasis Model Assessment
ICAM-1	Intracellular Adhesive Molecule-1
LDL	Low Density Lipoprotein (Cholesterol)
LERs	low energy reporters
mmHg	millimetres of Mercury
Mmol	Milimol
NHANES III	Third National Health and Nutrition Survey
Ors	Over-reporters
OVID	
PA	physical Activity
PAI-1	Plasminogen Activator Inhibitor Type 1
PPAR-α	Peroxisome Proliferator-Activated Receptor alpha
PPAR-γ	Peroxisome Proliferator-Activated Receptor gamma

PUFA	Polyunsaturated fatty Acids
PWBQ	Psychological Well-Being Questionnaire
RIA	Radioimmuno assay
RSS	Rosenberg Self-esteem Scale
SES	Socio Economic status
SPSS	Statistical Package for Social Sciences
UN	Urinary Nitrogen
UN/DN	urinary nitrogen/dietary nitrogen
UR	Underreporting
URs	Underreporters
UUN	urinary urea nitrogen
VLDL	Very Low Density Lipoprotein
VRs	Valid Reporters
WHO	World Health Organisation
Yrs	Years

SUMMARY

The present dissertation deals with broad issues of scientific importance to the field of Dietetics and Human Nutrition, crossing the frontiers which lie between what people say they eat, what they actually eat, what they should eat, and the ultimate effects of diet composition on health, which must be translated into advice and guidelines. Firstly it addresses veracity. Dietary mis-reporting and its relation with psychological and eating behaviour variables were assessed in adult women of varied BMI. A questionnaire, comprising several well-established psychological and eating behaviour tests and two questions to assess inclination to mis-report, was compiled and validated for understandability. Overall, 68% of participants declared an inclination to mis-report. Inclination to **under-report** was 29% among normal weight, 33% among overweight and 51% in the obese non-clinical groups; while the figure was 46% among the obese clinical patients. Among the same groups inclination to **over-report** were 39%, 29%, 11%, and 32%). Inclination to mis-report was associated with greater disinhibition, whether currently of dieting, frequency of dieting in the past, bingeing and dissatisfaction with body weight.

Dietary mis-reporting was then assessed in groups of men involved in food-related occupations (butchers and fishmongers) who hypothetically have better food skills than those involved in other occupations not related with food, i.e. schoolteachers. Food consumption was recorded prospectively during seven consecutive days (including weekends). Results showed an overall prevalence of 30% for dietary mis-reporting assessed by EI:BMR <1.2 with no significant differences between groups. The prevalence of EI:BMR <1.35 was 52% (butchers 78%, fishmongers 33% and schoolteachers 44%;

P=0.018). Dietary misreporting in these groups was associated with mis-reporting of alcohol consumption.

The eating habits and dietary practices of the same subjects involved in food related occupations were then assessed. The fact that these food skilled groups are potentially influential in other people's food choice, including that of their own families, invited the hypothesis that these groups may have healthier diets than other occupational groups. Indeed fishmongers reported a greater inclination to eat white and oil-rich fish than butchers. However, butchers did not report eating more meat. In this study, fishmongers also had a lower systolic blood pressure (130 ± 17 mmHg) than butchers (136 ± 11 mmHg) and schoolteachers (133 ± 15 mmHg). Knowledge and exposure to certain foods, such as fish, may contribute to the adoption of "healthier" patterns of food consumption among some subgroups. This result, in broad agreement with the limited literature on fish consumption, stimulated a controlled trial of fish consumption.

The third study was therefore designed to assess the effects of salmon consumption on vascular parameters of the metabolic syndrome. The opportunity was used to study the effect of salmon on serum leptin as well, following a report of lower leptin concentrations amongst fish-eaters, suggesting increased leptin sensitivity. Forty-eight healthy, non-obese men and women consumed 125g salmon daily for four-weeks followed by a 4-week control period without fish. Fasting lipids, adiponectin, leptin, insulin, fibrinogen and ICAM, as well as blood pressure were assessed before and after each intervention period.

An overall good compliance for the consumption of fish was confirmed by significant increases, compared with no-fish and baseline values, in plasma EPA (mean +96 μmol/)

and DHA (+75 μmol/L). On average a significantly lower systolic (-4 mmHg) and diastolic (-3 mmHg) blood pressure, triglycerides (-0.13 mmol/l), VLDL (-0.07 mml/l), and LDL (-0.17 mmol/l), and higher HDL (+0.09 mmol/l) were observed with fish consumption versus no-fish (all $p<0.05$). Significant decreases with fish were also observed in the total cholesterol/HDL ratio as well as LDL/HDL. These changes with salmon intake predict around a 25% reduction in CHD risk. A significant increase from baseline in adiponectin levels was observed with fish consumption (+1.21 μg/ml/1000, $P<0.05$). The increase in adiponectin during no-fish periods following salmon consumption (+0.87 μg/ml/1000) achieved a p-value of 0.086. These results were independent of weight change. These data are very suggestive of an effect of salmon consumption increasing adiponectin.

Within this study of fish consumption three different manoeuvres were explored as possible ways to identify and exclude bias associated with veracity. On average, plasma EPA rose by 96 μmol/ml but 6 subjects showed minimal changes (< 20μmol/ml). Eleven subjects had reported EI <1.2 x BMR and 16 subjects reported an intentional inclination to misreport. The effects of daily salmon consumption on indices of metabolic syndrome were examined after excluding subjects. Results showed the same patterns described above. Dietary mis-reporters complied with fish consumption and therefore were benefited in their metabolic profile in the same way as acceptable reporters. Statistical significance was not affected because of the smaller sample size of the group.

The results of this series of studies illuminates one of the darkest corners of dietetics, and one which has often been ignored when using data from individual dietary assessments or from large scale surveys of food consumption. One conclusion is that it would seem useful

to ask people who are undergoing dietary intake estimation about their inclination to misreport. The common practice of eliminating data from subjects with implausibly low intakes should be matched with equal scrutiny for over-reporting. The problem appears to be present in those who are underweight, normal weight and over weight, and in those reporting low, medium and high intakes (with different contributions from over and under-reporting). The studies in this dissertation do not necessarily provide representative population-based samples, so extrapolation of quantitative analysis to other groups needs these studies in the future.

The concept that people with food-related occupations may have different attitudes to food and eating was supported to some degree by these results, and these data are interesting in suggesting that fishmongers may eat more fish, and that this have benefits in protecting against metabolic syndrome. Fishmongers are a declining occupation as supermarkets take over, and their potential for promoting healthy eating may have been lost in UK.

The work of this thesis contributes new information which might help in future dietary guidelines with respect to fish and the prevention of metabolic syndrome and CHD, which are especially relevant to populations where rising waist circumferences and obesity mean that this problem is set to become even more prevalent. Daily fish consumption appears to offer advantages. For many people this is acceptable. For the majority, however, future work will need to evaluate smaller amounts over longer periods and to identify the specific components – maybe fish oils – responsible.

Chapter 1

Challenges in human nutrition and dietetics: Dietary mis-reporting - a literature review

1.1 INTRODUCTION

At the core of public health nutrition is a need to establish what people eat and to relate this to health indices, and then to define guidelines which can be expected to improve health. Large numbers of assumptions are commonly made about exposure to food and nutrients and about what constitutes the "habitual diet" of individuals. For some health outcomes this may in fact not be the appropriate measure (e.g. cancers which might be promoted by occasional dietary toxins). There are also genetic factors which might mean that different diet composition suit different people. These issues are beyond the scope of the present thesis, whose starting point is the problem of veracity in food behaviour.

This chapter reviews the literature available on dietary assessment and mis-reporting in different subgroups of the population, exploring its prevalence and its determinants.

1.1.1 Importance of food consumption information

Diet and nutrition are key factors in promoting and maintaining good health throughout life. A poor diet is a well-established determinant of chronic non-communicable diseases and malnutrition (WHO 2003). The patterns of food consumption form a dynamic process; several factors influence the food choice and preferences of individuals and populations.

These were summarised by Rozin and Vollmecke (1986) and Booth (1990).

1. ***Biological*** Physiological, pathophysiological, genetic, age, sex

2. ***Socio-cultural*** groups of origin (e.g. regional provenance, nationality) group norms, social manners, social stratum, cuisines

3. ***Psychological*** Emotions, motives, attitudes, perception, thinking, learning,

4. ***Socio-economic*** Prices, income, marketing, availability

5. ***Anthropological*** Definition of what is edible, symbolic meaning of foodstuffs, concept of food, religion, food taboos, foods customs and beliefs, spirituality/religion

Other factors govern the food availability. Although most of these are not limiting factors in most western countries, they are still influential in developing societies, e.g. agricultural practices, climate and ecology.

Information on food consumption is of paramount importance for the science of nutrition as well as other related fields. Indeed this information forms the cornerstone of nutrition science and day-to-day dietetic practice. Some of the uses or applications of information on food consumption are listed below:

1. ***Assessing the nutritional status*** (What we eat, what we are and what we can do)

2. ***Define dietary allowances*** (DRVs)

3. ***Designing dietary guidelines and monitoring their achievement***

4. ***Establishing dietary and nutritional risk of individuals and populations***

5. ***Establishing and quantifying nutritional and dietary factors related to disease***

6. ***Designing food and nutrition assistance programmes***

7. ***Providing nutrition education and guiding for food choice***

8. ***Planning national food security.***

1.1.2 Measuring food consumption: Its problems

The true "habitual" food consumption of an individual or a population is the information usually sought to address most of the issues listed above. Various methods for measuring individual's food intake are available including diet records to assess prospective food intake, recalls, food frequency questionnaires and dietary histories which all assess food consumption retrospectively. Concern about the advantages and disadvantages of such methods, has usually drawn considerable attention. All retrospective methods depend on memory and some probe memory over a prolonged period. Several sources of error, inherent to the different dietary methods, which all depend on the memory and recording skills as well as co-operation of subjects, are known to influence the validity of food consumption data (Bingham, 1987, Black & Cole 2001). "Optimistic bias" is the term used to describe bias towards the perceived ideal- that may be unintentional (Shepherd, 1999). However there is also evidence suggesting that biased dietary information might be the result of conscious alterations on what people eat and/or on the information they self-report. This is exemplified by certain population subgroups adopting apparently extreme and untenable beliefs about their food consumption, for example obese subjects (Buhl *et al.*, 1995). Therefore it is not surprising that what seems to be a simple task it

has been recognised as one of the most difficult ones (Garrow, 1988) and the possibility of having a dietary method free of error or bias in the future seems rather improbable (Beaton, 1994).

1.1.3 Validating food consumption data: Identification of mis-reporting

There is no independent direct ways to check, or validate, the food choices by free-living subjects. Based on the premise that the pattern of modern trash might be used as an indicator of human consumption, household refuse analysis was used to validate reported food consumption in the University of Arizona Garbage Study (Ritenbaugh & Harrison, 1984). Strikingly, twenty years ago, this study described the systematic mis-reporting of food consumption: this inclined **over-reporting** of culturally approved, healthful behaviours (whole grains, fruits, vegetables, skim milk); and **under-reporting** of culturally undervalued, 'indulgent' behaviours (alcohol consumption, high fat snacks, sugared cereals, cookies). It cannot be ascertained whether this under-reporting was intentional, or if it represented "optimistic bias".

Validation studies on food consumption data, against an objective reference method, usually a biological marker, have shown that information on food consumption is of dubious value in almost any diet study. The celebrated apparent inverse association between energy intake and body mass index is a common finding to most diet studies, as an example of such a problem (Romieu at al., 1988; Prentice and Jebb, 1995). It was considered by Keen *et al.* (1979) to represent a mysterious metabolic adaptation to obesity for which they coined the term "low energy throughput". Studies of energy

expenditure have proved that obese people must in fact eat more to avoid weight loss (Prentice *et al.*, 1986).

Mis-reporting, i.e. implausible food intakes for a reference physiological measure such as total energy expenditure or urinary nitrogen, is now recognised an important source of error in food consumption data. The availability of methodology to assess energy expenditure and urinary nitrogen, under neutral energy balance conditions, has allowed the identification of energy and protein mis-reporting (Prentice *et al.*, 1986; Bingham and Cummings, 1985). Under-reporting of energy intake, whether given by under-recording (the ineffective recording or recalling of foods) or by under-eating (resulting in a consequent weight loss during the recording period which sometimes may be too short to demonstrate weight change) has been shown to be more significant than over-reporting (Johansson *et al.*, 1998; Blundell 2000).

Porikos *et al.* (1977) used secret cameras and automated vending machines to establish that obese subjects simply failed to record some of the foods they ate and recorded prospectively.

The use of different methodologies makes comparisons between studies difficult. Different methodologies have been used: weighed records or estimated food diaries of a different length, food frequency questionnaires, dietary history and 24-hour recall. Energy expenditure has been assessed by means of the double labelled water (DLW) method, by energy balance techniques, or from basal metabolic rate or resting metabolic rate, either measured by gas exchange or estimated, multiplied by a physical activity score.

The lack of other biomarkers has made it difficult to examine the extent of mis-reporting of any other nutrient; however this is an area of research receiving more attention and the development of new biomarkers for the validation of different foods and nutrients is expected to happen in the next future. Intentional mis-reporting has not previously been researched. Over-reporting of food intake is clearly a feature of "anorexia nervosa". The reasons why normal people mis-report food consumption however have been explored only recently and the available literature is scarce. Nevertheless there is evidence suggesting that the process of reporting of food intake is influenced by different factors e.g. social and/or psychological factors. In 1984 Worsley *et al.* reported an effect of social desirability on the reporting process of food intake. More recent studies by Taren *et al.* (1999) and Hebert *et al.* (1997) have confirmed these findings.

The current position prior to this thesis is that dietary mis-reporting is considered a common problem amongst the obese, and suggestions of systematic mis-reporting suggest a conscious, intentional component rather than lack of perception regarding portion size. No study has actually asked people whether they would be inclined to mis-report their food consumption.

1.2 A REVIEW OF THE LITERATURE

1.2.1 Methods

A review of the literature was carried out in order to identify the articles dealing with the measurement of FI and total energy expenditure (TEE) and/or BMR and reporting results in terms of FI under-reporting. Those were searched by using MEDLINE,

PUBMED and OVID, electronic databases and crosschecked with the references cited in the sources identified.

The key words used in the search were: mis-reporting, under-reporting, under-eating, under-recording, EI:BMR, energy balance, obesity, food intake, energy intake, macronutrients intake, meals, snacks, post-obese.

The articles included in the present work are all those identifying and informing the degree of mis-reporting of energy and/or macronutrient intake, and/or the prevalence of mis-reporters. Once identified, for comparison purposes, the articles were classified according to age group. Comparisons, when possible, were made in terms of gender and body mass. The determinants or correlates of mis-reporting, when available, were also identified in these studies.

1.2.2 Dietary mis-reporting in children and adolescents

Eighteen studies carried out in children and/or adolescents are listed in **table 1.1**. Methods to define under-reporting have usually employed DLW for energy expenditure and different dietary methods.

Obese versus non-obese Studies comparing obese and non-obese children (9-15 yrs) show significantly more under-reporting of energy intake in the obese that may under-report energy intake up to 40%. Under-reporting is not significantly different between genders in both obese and normal weight. In terms of age, dietary under-reporting seems to be low (8 to 10% of energy intake) in children under 8 years of age; slight over-reporting of energy intake seems to be more common among this group. Of exceptional contrast is the study by the Kaskoun *et al.* (1994) who reported a substantial over-

reporting of energy intake in children aged 4 to 10 years. These authors suggest the use of a FFQ with serving sizes typical of adult subjects as a possible explanation of the differences observed. No other study in the literature has reported comparable results so far. Under-reporting increases up to 20% of EI in children aged 8 years and older. There seems to be a difference between ethnic groups, with African-American children under-reporting more than white children (Champagne *et al.*, 1996; 1998). Differences regarding educational status between these groups might be a possible explanation to such discrepancies.

Dietary methodology Two studies seem to provide support in favour of the use of dietary histories in normal-weight children (Livingstone *et al.*, 1992; Maffeis *et al.*, 1994). The first study reported a certain bias toward over-reporting. In the second study the authors concluded that '... dietary record and the diet history are not valid means of assessing energy intake in obese pre-pubertal children'. However it is a fact that the diet history showed agreement with food records and energy expenditure as determined by DLW in non-obese children; while in obese the discrepancy with DLW was smaller than that by FR (see **table 1.1**).

Prevalence of mis-reporters The prevalence of mis-reporters is not addressed in any of the studies analysed.

Selective mis-reporting of nutrients Selective mis-reporting of specific nutrients has not been evaluated in these age groups. However some studies suggest the possibility for selective mis-reporting of fat and carbohydrates. Champagne *et al.* (1998) found that the reported proportion of fat and carbohydrates (possibly due mainly to non-milk extrinsic

sugars e.g. soft drinks) of 118 subjects was 23% below the average intakes for the American population. However on statistical analysis there was no difference. This finding seemed more prominent among girls, particularly African-American girls.

Determinants of dietary mis-reporting Among children, body mass is the strongest determinant of mis-reporting. A higher body mass index and or body fat is positively associated with reporting bias. These studies also provide evidence for differences in mis-reporting amongst different racial groups, with African-American subjects mis-reporting more significantly than their white counterparts ($P<0.05$). Cultural differences between these groups seem to be an attractive research issue and are important to keep in mind when assessing food intake in different ethnic groups. Age, particularly after 8 years of age, is another variable positively related to dietary mis-reporting in this group.

Effects of ethnicity In children some studies have reported no differences related to ethnicity (Kaskoun *et al.*, 1994). However only a small group of Mohawk children (N=9) was included in this study. On the other hand Champagne *et al.* (1998) comparing white and African-American children, found that the latter group under-reported significantly more of their FI in relation with their white counterparts (**see table 1.1**). When compared by gender, only the difference in mis-reporting between boys reached statistical significance (African-American -26.2±3.1, White –16.8±3.2, $P<0.05$). Girls had similar frequencies (Africa-American 26%; white 28%) This study also showed patterns of increasing mis-reporting related to age, which also have been reported by Bandini *et al.* (1997).

Remarks At early ages, factors such as memory, attention and perception represent the main problems in assessing food consumption in school-age children. However the fact that an adult may provide help in reporting food intake of children does not seem to guarantee a lower bias possibly related to their own eating behaviour. Dietary mis-reporting is present in childhood at an age in which children become more aware of their eating habits. It increases with age, and gender does not seem to be a factor of difference. From the studies available it seems that after the age of 8 years dietary mis-reporting is consistently present and related to body mass just as in the case of adults. Despite an improvement in cognitive abilities to estimate their food consumed, this seems to be overridden by psychological and social pressures particularly related to body mass and body image. As children enter adolescence these pressures are stronger. What children and adolescents report they eat cannot be equated simply with what they usually do.

1.2.3 Dietary mis-reporting in adult obese/overweight subjects

Thirty-two studies assessing dietary mis-reporting in obese and overweight (i.e. BMI ≥25) adults (~18-67 yrs) showed that obese and overweight subjects are more likely to under-report food intake than non-obese. The magnitude of energy intake under-reported by this group varies in the range of 20 to 40% but may be more prominent (50-60%) among obese unsuccessful dieters (**table 1.2**).

Prevalence of mis-reporters The proportion of individuals identified as under-reporters among the obese goes from 40 to 60%, with a higher proportion among unsuccessful dieters. No reference to over-reporting EI or prevalence of over-reporters is mentioned

in these studies. No significant differences in energy intake under-reporting between obese men and women are observed on these studies.

Effects of dietary methodology Two studies (Heerstrass *et al.*, 1998; Kroke *et al.*, 1999) employed a FFQ and a number of 24-hr recalls to assess food intake; compared to the doubly labelled water method and the EI:BMR ratio. The FFQ resulted in less under-reporting assessed as a higher EI:BMR ratio than 24-hr-recalls.

In contrast, Fricker *et al.* (1989) by means of a dietary history reported no mis-reporting in both men and women. This was an exhaustively long dietary history. It seems possible that asking more questions produces a greater apparent food intake but not necessarily a more accurate one.

Selective nutrient mis-reporting In relation to macronutrient mis-reporting, the lack of accurate biomarkers, with the exception of protein, makes difficult to establish whether obese subjects truly consume high amounts of fat and/or carbohydrates as well as whether mis-reporting could be selective for these nutrients. The studies assessing protein intake by means of urinary nitrogen (UN) assessment do not show that there is a systematic pattern for mis-reporting of protein intake (Heitmann 1993; Heitmann and Lissner, 1995; Lindroos *et al.*, 1993, 1999). Recently, Goris *et al.* (2000) reported a selective pattern of fat under-reporting. Such a finding was derived from the interpretation of dietary data by regression analysis. Errors due to variability in food composition, e.g. portion size, are a factor to bear in mind when interpreting these results.

Determinants of dietary mis-reporting Most studies reported an association between mis-reporting and different indices of body mass and/or any of its components. However, several studies searching for behavioural and/or psychological determinants show that people dieting mis-reported energy intake more than non-dieters independently of age and gender (Heitmann 1993). Another study, (Johnson *et al.*, 1998) reported a high percentage of body fat and poor literacy, assessed by means of the Wide Range Achievement Test, as the best predictors of energy intake mis-reporting (r=0.52). More recently Taren *et al.* (1999) showed that *social desirability*, behind age and percent body fat, was the best psychological factor predicting mis-reporting (P=0.05). In addition, in this latter study body image concern, body dissatisfaction and identification of body silhouettes as healthy were also associated with mis-reporting. One study (Poppitt *et al.*, 1998) showed restrained-eating subjects as the most prominent group under-reporting energy intake.

Remarks Some similarities between the studies reviewed are worth a mention. Amongst the studies assessing obese subjects, those which included subjects refractory or resistant to dietary treatment with low calorie diets (Buhl *et al.*, 1995; Lichtman *et al.*, 1992) showed the largest discrepancies in the reporting of food consumption. How should we interpret the results of those studies? Were the studies showing low mis-reporting carried out in highly motivated obese subjects? Did the studies showing a high mis-reporting rate include a high proportion of 'resistant' obese subjects? Unfortunately no data are available to answer these questions.

Three findings deserve mention and further study. Firstly in the study by Weber *et al.* (2001) the use of a different computerised nutrition program contributed to a

discrepancy going from 4-12 % when expressing the mis-reporting of FI as a percentage. Although food composition tables have been indicated as a source of error (Bingham, 1987), its effects in dietary mis-reporting were unknown.

Secondly, Goris *et al.* (2000) showed that mis-reporting by obese subjects was due mostly to under-eating (two thirds of the energy mis-reported). Only one third of the energy mis-reported was explained as under-recording in that study. The only fact of recording FI could lead to the subject to modify its usual diet in order to facilitate the process of recording or weighing of foods (Bingham, 1987). Whether this adaptation is a response to awareness of what those subjects were previously eating, or an attempt to comply with a more socially or clinically desirable image in the case of obese subjects is unknown (Blundell, 2000). The selective under-reporting of fat intake reported by those authors should be taken cautiously. In addition only four subjects reported FI within ± 20% of their energy requirements (energy expenditure) and their intakes of fat ranged from 35% to 50%, thus showing a wide variation.

Finally, the study by Fricker *et al.* (1989) deserves a special attention. This is not only because of the finding of no mis-reporting in their sample of obese men and women, but because it shows that a long questionnaire (DH, 450 items) could be successfully applied and overcome some of the pitfalls in dietary assessment. However most of the studies are characterised by the search of a more simplified, and less time-consuming and burdensome method, which by no means guarantee the validity required.

1.2.4 Dietary mis-reporting in adult normal-weight subjects

Table 1.3 list twenty-eight small studies carried out in adult (19-64 yrs.) normal weight subjects (i.e. BMI from 18.5 to 24.9), restrained-eating subjects, dietitians and athletes. The proportion of energy intake under-reported goes from 10 to 20%; only one study in female distance runners under-reported EI above 30%.

Gender differences in dietary mis-reporting Differences between men and women are difficult to draw from these studies given that only three studies included men. One showed a slightly higher under-reporting in men than in women and two showed no difference. Martin *et al.* (1996) identified 34.5% of under-reporters in their study, however Mertz *et al.* (1991) in a series of studies using energy balance techniques reported that up to 82% of their sample might have under-reported their food consumption; approximately 10% of this study's sample was identified as over-reporters. The only study on post-obese subjects showed results similar to other women studies.

Determinants of dietary mis-reporting Among the possible determinants of dietary mis-reporting, Ortega *et al.* (1996) found that weight conscious subjects under-reported more their EI than non-conscious subjects. In addition, Goris and Westerterp (1999; 2000) reported that under-eating accounted for total EI under-reporting (i.e. no under-recording of foods was observed) in female dietitians; the same dietitians reported unbiased dietary information in a second study. These authors concluded that under-reporting was not necessarily the result of a dishonest action. The authors suggest that confronting subjects with their results could eliminate mis-reporting. Although this strategy could be a way to overcome mis-reporting in highly motivated subjects, it is

impractical. It is not possible to tell whether the EI reported during the second study was representative of the usual, or whether it was a response influenced by seeking social approval (reputation as dietitians).

In addition, a study comparing restrained and non-restrained subjects (Tuschl, *et al.*, 1990) showed only slight over-reporting of EI by restrained subjects (i.e. no under-reporting); however in relation to the proportion of macronutrients a significant difference was observed in carbohydrate, fat and alcohol consumption.

Another study (Muhlheim *et al.*, 1998) used the "bogus pipeline procedure" (an experimental procedure leading the participants to believe that an experimenter could verify (pipeline) their responses and thus motivating them to report more accurately) in an attempt to obtain reliable information on EI using the DLW method as the model of validation. Both groups control and experimental groups still under-reported their EI (control group -48% Vs. -52%; experimental group -45% Vs -39%). Although these improvements reached statistical significance, the fact is that the subjects were still under-reporting substantially.

The study by Black *et al.* (1995) in post-obese subjects shows still high rate of under-reporting. When assessing restraint, these authors found marked differences between the subjects. The considered mis-reporters were classified as restrained, while the valid reporters showed a milder degree of restraint. Separating the subjects on this basis, mis-reporters under-reported EI by -41.8%, while the valid reporters did it only by -10.2%.

Dietary methodology Sawaya *et al.* (1996) compared a FFQ, 7-d WR and a 24-hr recall to assess food consumption in a group of women. Using the DLW method, these authors

found similar under-reporting of EI with the FFQ and 7-dWR but considerably less with the 24-hr recall. Black *et al.* (2000), used DLW and urinary nitrogen (UN) to compare four 4-dWR with a dietary history. Energy and protein intakes were more accurate with a DH than WR in this study. Bathalon *et al.* (2000) assessed food intakes in restrained and non-restrained eaters. Using the DLW methods these authors found that subjects in both groups reported more accurately with 7-dWR than FFQ or 24-h recalls. Adjustments for weight changes during study reduced the differences observed among non-restrained but 7-dWR were still the best dietary assessment method in restrained subjects.

Remarks The studies listed in **table 1.3** confirm that mis-reporting is not a phenomenon characteristic of any particular group of people, e.g. obese. People likely to have a better access to nutrition information such as athletes (Edwards *et al.*, 1993; Hill & Davies, 1999; Beidleman *et al.*, 1995) and dietitians (Goris & Westerterp, 1999; 2000) mis-report their FI when asked to record it. This is surprising because apart of nutrition knowledge, high academic or educational level, dietitians are commonly the people in charge of training subjects for reporting FI in most dietary studies. With no doubt this makes the panorama to look hopeless.

1.2.5 Dietary mis-reporting in older people

Thirteen studies in older people are listed in **table 1.4**. Under-reporting of energy intake among this group goes from 20-30% for women and from 12 to 14% for men. Only the studies by Tomoyasu *et al.* (2000) and Black *et al.* (2000) have reported lower figures

for mis-reporting in women. No comparisons between obese and normal-weight are provided in these studies.

Selective mis-reporting of nutrients In relation to the mis-reporting of macronutrients, Bingham *et al.* (1995) reported that based on the urinary nitrogen to dietary nitrogen ratio (UN/DN), mis-reporters (top quintile) were heavier and showed a lower EI:BMR ratio. Visser *et al.* (1995) reported a negative difference of 13 % between the DN and UN. On the other hand, data from Rothenberg (1994) shows a clear pattern of DN over-reporting when using a FFQ to assess food intake compared with the data from food records. Another study on restrained versus non-restrained women (Bathalon *et al.*, 2000) showed that restrained eaters reported significantly higher intakes of protein ($P \leq 0.05$) and lower fat ($P < 0.01$).

Remarks In general, data from studies carried out in older subjects do not show difference from those in other age groups. However the importance of addressing mis-reporting in this group resides in their willingness to participate in dietary studies compared with younger subjects.

1.2.6 Dietary mis-reporting in population-based studies

Thirty-five population-based studies are compared in **table 1.5**. The method used to assess energy mis-reporting in population studies is the energy intake to basal metabolic rate ratio (EI:BMR) multiplied by a physical activity (PA) score. This method allows the estimation of the prevalence of mis-reporters and comparisons between populations. A lack of information about the PA level of the sample implies the use of different cut-off points to estimate the prevalence of mis-reporters.

Prevalence of mis-reporters The prevalence of under-reporters ranges from 10 to 40% in men (mean 15%) and from 20 to 50% in women (mean 28%). In obese and overweight subjects these figures rose to around 40% in men and 50-60% in women. Contrasting with the rest of the studies Lafay *et al.* (1997) showed a higher prevalence of male than female mis-reporters.

A Finnish survey (Fogelhom *et al.*, 1996: Hirvonen *et al.*, 1997) showed an increase in the prevalence of mis-reporters between 1982 and 1992. Fogelhom *et al.* (1996) provided evidence supporting a more prominent increase in food consumption than a decrease in physical activity as determinants of obesity during that period, thus supporting a true increase in dietary mis-reporting.

Selective mis-reporting of nutrients Population studies show that intake of under-reporters and over-reporters differ from the considered "valid reporters". Consistently, under-reporters refer a higher intake of protein and/or CHO and lower fat than valid reporters. It is uncertain whether these findings truly indicate a selective under-recording of dietary fat, which might be anticipated if low-fat diets were considered "desirable" by subjects.

Determinants of dietary mis-reporting Several factors have been related to mis-reporting in populations studies. Results from the NHANES III study (Briefel *et al.*, 1997) showed age as a significant determinant of mis-reporting ($P<0.01$); mis-reporters were on average 5 and 4 years older than male and female "valid reporters", respectively. Education was another factor, with more low-educated (0-8 yr) men and women and less high-educated (≥13 years) men among mis-reporters ($P<0.01$). In addition, a BMI ≥27.8

for men and ≥27.3 for women, high rate of dieting (40% of males and 50% of females), low socio-economic status (SES) and a higher proportion of non-Hispanic black people among men (P<0.01.) were also related to mis-reporting.

Price *et al.* (1997) in a British national survey found significant associations between low social class, low educational achievement and lack of remuneration with a lower report of energy intake in women, but not in men. Similarly, Stallone *et al.* (1997) found an association between socio-economic status and under-reporting in the Whitehall II study, in which the prevalence of under-reporters increased among lower employment grades.

In a survey by the Department of Agriculture from the U.S.A. (Krebs-Smith *et al.*, 2000), mis-reporters were characterised by higher proportions of females, older aged, non-Hispanic blacks, lower educational levels, and lower income subjects than non-mis-reporters subjects (P<0.01).

Remarks The use of a BMR x physical activity multiple has allowed the assessment of diet and mis-reporting in large samples. However the lack of proper information on the actual PA levels of these populations has caused the use of different cut-off points. The studies summarised in **table 1.3** show that depending on the cut-off point chosen for the EI:BMR ratio, the proportion of mis-reporters varied from 20 to 30 % when a cut-off point ≥1.1 was used, to a range of 35-50% when a higher cut-off point was chosen. The proportion of mis-reporters increased importantly with greater BMI, with at least half of the subjects classified as overweight and obese groups being under-reporters.

Hirvonen *et al.* (1997) showed that the inclusion or exclusion of under-reporters did not affect the macronutrient proportion but it did affect the micronutrient intakes. A possible

explanation for this is that even when these authors excluded the under-reporters, the sample could have still contained under-reporters who were not identified due to higher levels of PA than those considered by the cut-off point used for the EI:BMR ratio. The effect of the presence of over-reporters is another issue to keep in mind since little attention has been given to this class of mis-reporting.

1.3 Conclusions

The dietary mis-reporting dilemma described as "the human nutritionist's guilty secret" (Garrow, 1995) has been acknowledged also as an enigma for researchers on other fields, e.g. psychology (Blundell, 2000). There seem to be many reasons for this problem. The data analysed here suggests that there is not yet a "gold-standard" way to assess true dietary intake accurately. Under-eating seems to be more common in both, small laboratory studies and as reflected by low EI:BMR ratios in large surveys.

The tendency to report low energy intakes, sometimes implausibly so, has become a common feature in dietary studies. Although many studies have shown an association between mis-reporting food intake and physical characteristics such as body weight and body fat, those are only associations not implying cause. Mis-reporting has been also related to socioeconomic status, a higher influence of feelings, hedonistic beliefs for food, and other conditioning individual behaviours and/or psychological factors. The study and understanding of mis-reporting food intake is not a problem particular of the science of nutrition, as Blundell has pointed out (Blundell, 2000) it is also a problem for psychology; however it looks as though the problem goes beyond the scope of those two disciplines. So it is sensible to search aspects of psychological nature, but also of

Table 1.1. Studies assessing misreporting in children and adolescent: Comparisons based on body mass, gender and age

Source/ Country	N	BMI (Kg/m^2)	Age (y)	Methods	EI Misreporting (%)	Mis-reporters Prevalence (%)	Macronutrients Misreporting (%)	Mis-reporting Determinants /Comments
Non-obese/ Obese								
Bandini *et al.*, 1990 USA	N-Obe 14 ♀ / 14 ♂ Obese 13 ♀ / 14 ♂	≈21 ≈35	14±1 15±2	14-d FR + DLW + EB	N-Obe -19 / Obe -41% (P<.0001) ♀ -34 ♂ -27% (P<.05)	---	---	---
Maffeis *et al.*, 1994 Italy	N-Obe 6 ♀ / 6 ♂ Obese 6 ♀ / 6 ♂	16±2 /16±1 25±4 /24±2	9±1	3-d WR + DH Heart rate 3-4d	WR ♀&♂ N-obe -5 Obe -28% DH N-obe ♀+5 / ♂+11% Obe ♀ -8 / ♂-20%	—	---	Body Weight % Body Fat
Champagne *et al.*, 1998 USA	Afric-Am 27 ♀ / 29 ♂ White 31 ♀ / 31 ♂	 18±1 / 21±1 19±1 / 20±1	10	8-d FR DLW	Af. ♀/♂ -26/ -28 White -24/ -17% Obese -25/ Lean -21; fat distrib: Central -32 Peripheral -17%	---	No-significant diff. with NHANES III	Gender Ethnicity
McGloin *et al.*, 2002 UK	114 ♀ & ♂ Risk of obesity: low/ high/ Obese	Low 15±2 High 17±2 Obese 23±2	7±1 7±1 7±1	7-d WR DLW	Low risk -1.6% High risk -4.7% Obese -13.7%	---	---	% Body Fat
Female/male								
Livingstone *et al.*, 1992 Ireland	41 ♀ 37 ♂		3-18	7-d WR (7-18 y) + DH (3-18 y) DLW	7-dWR ♀ ♂ DH ♀ ♂	—	---	---
		(3y) 16/			-- -- +15% +9%			
		(5y) 15			-- -- -3% +24%			
		(7y) 16			+24% -8% +22% +4%			
		(9y) 17			+7% +1% +2% +10%			
		(12y) 19			-8% -15% +7% +20%			
		(15y) 20			-12% -32% +10% -9%			
		(18y) 25			-32% -23% -6% +1%			

Source/ Country	N	BMI (Kg/m^2)	Age (y)	Methods	EI Misreporting (%)	Mis-reporters Prevalence (%)	Macronutrients Misreporting (%)	Mis-reporting Determinants /Comments
Kaskoun *et al.*, 1994 USA	23 ♀ 22 ♂ White/Mohawk	≈17/ ≈16	4-7 y	Short FFQ+ DLW	♀ +62% ♂ +5.2%	—	---	—
Champagne *et al.*, 1996 USA	Afric-Am. 5 ♀ 6 ♂ White 6 ♀ 6 ♂	21±2 / 23±2 19±2 / 18±2	11	8-d FR DLW	Afric-Am -37% (≈♀-33, ♂-40) Whites -13% (≈♀-12, ♂-15)	—	—	—
Johnson *et al.*, 1996 USA	12 ♀ 12 ♂	18±3 / 18±3	6±1	3 multi-pass/ 24-hR + DLW	♂ -10 % ♀ +4.1%	—	—	—
Bratteby *et al.*, 1998 Sweden	25 ♀ 25 ♂	21±3 20±3	15	7-d FR + DLW	♀ -22±16% ♂ -18±18%	—	—	—
Ambler *et al.*, 1998 USA	32 ♀ 39 ♂ Ethnic. Mixed 5-wk 2-h training Vs control	—	15-17	3-d FR 24-h recall DLW	Control Trained ♀ 0.0% -26.6% ♂ 0.0% -10.6%	—	---	
Lindquist *et al.*, 2000 USA	13 ♀ 17 ♂ Ethnically mixed	—	10±1	3-taped FR+ 3 24-hR+ DLW	♀ -14.4% ♂ -0.5%	URs/ORs/VRs FR 61/ 26/ 40% 24-hR43/ 40/ 17%	—	Fat mass Age
Fisher *et al.*, 2000 USA	73 ♀ 76 ♂ Ethnically mixed	18-39	8±2	2-3 multi-pass 24-hR + DLW	Overall +14%	URs/ ORs /VRs 21/ 46/ 32%	—	
O'Connor *et al.*, 2001 Australia	25 ♀ 22 ♂	17±2 16±2	6-9	3-d FR + DLW	♀ +5 ± 24% ♂ +4 ± 23%	—	—	
Sjoberg *et al.*, 2003 Sweden	17 ♀ 18 ♂		16	DH + DLW	♀ -18% ♂ -7%	URs/ ORs /VRs 26/ 17/ 57%	—	

Source/ Country	N	BMI (Kg/m^2)	Age (y)	Methods	EI Misreporting (%)	Mis-reporters Prevalence (%)	Macronutrients Misreporting (%)	Mis-reporting Determinants /Comments
Females by age								
Davies *et al.*, 1994 UK	39 ♀ 42 ♂	---	<4.5	4-dWR + DLW	Overall= -3.1% / 1.5-2.4 yr=- 6% 2.5-3.4 yr= –5% 3.5-4.4 yr= +0.7%	---	---	---
Bandini *et al.*, 1997 USA	109 ♀ Pre-menarch	—	8-16	14-d FR + DLW	All -12% /(8y)-3% /(9y)-5% /(10y)-16% /(11y)-19% /(12-16y)-22%	—	—	TEE Age
Bandini *et al.*, 2003 USA	26 ♀ Longitudinal study: age 10, 12, 15	17/ 19/ 22	10	FFQ DLW	10 yr old –12% / 12 yr old -23% 15 yr old -32%	—	—	
Vegan/Omnivores								
Larsson *et al.*, 2002 Sweden	(♀ & ♂) 16 Vegan 16 omnivores	V♀ 25/ ♂ 22 O♀ 20/♂ 22	17±1	DH ÷ DLW ÷ UN	Vegan -14% Omnivores -12%	---	Protein 0.0%	---

♀= women; ♂=men; 4-d WR four-day weighed record; DLW= doubly labeled water method; FFQ= food frequency questionnaire; 7-d WR= seven-day weighed record; DH= dietary history; DEXA= dual energy x-ray absorptiometry; EB= energy balance techniques; TEE= total energy expenditure; CHO= carbohydrate; BMRm= basal metabolic rate measured; ; 7-d FR= seven-day food record

Table 1.2 Studies in obese/overweight adult subjects: Comparisons for energy intake and/or dietary protein mis-reporting according to body mass overweight and gender

Source/ Country	n	BMI (Kg/m2)	Age (y)	Methods	Energy Intake Misreporting (%)	Mis-reporters Prevalence (%)	Macronutrient Misreporting	Misreporting Determinants/ Comments
Non-obese/ Obese								
Prentice *et al.*, 1986 UK	22 ♀	13 n-obe 22±2 9 Obe 33±5	29±5 35±5	7-d WR + DLW BMRMeasured	Non-Obese -2% Obese -33%	URs 27.3%	---	---
Heitmann, 1993 Denmark	201 ♀ 199 ♂	---	35, 45, 55, & 65	DH + UN + Bioimpedance	---	---	Protein by Body fat tertile Low Mid Upper ♀ -23 -18 -13 ♂ -21 -8 -10	-% Body Fat -Slimming
Westerterp *et al.*, 1992 Netherlands	19 ♀/18 ♂	Non-Obese 20±1 / 24±2 Obese 31±3 / ♂32±4	22-62	7-d FD DLW	Non-Obese -11±15% Obese -30±18%	URs BMI<25 54.5% BMI 25-30 89% BMI >30 83%	---	BMI
Lindroos *et al.*, 1993 Sweden	64 ♀ & ♂	no-obe 19-26 Obese 27-49	39±10 46±11	4-d FD + DH+ UN+ BMRMeasured	DH No-obe +5 Obe +4% 4-d FR No-obe +3 Obe -23%	---	Protein DH No-obe -3 Obe+16 FR No-obe -12 Obe -14	---
Poppit *et al.*, 1998 UK	33 ♀	No-Ob 24±3 Obe 41±8	47±14 38±13	24-hR + Observed EI+ BMREstimated EI/ BMR =1.4	Obe -13 No-Obe -12% Restrained -19.1% Non-restrained -10.8%	---	All Obese CHO -19.9 -23.7 Add sugar -20.1 -25.9 E-snacks -35.8 -40.6	---

Source/ Country	n	BMI (Kg/m2)	Age (y)	Methods	Energy Intake Misreporting (%)	Mis-reporters Prevalence (%)	Macronutrient Misreporting	Misreporting Determinants/ Comments
Heerstrass *et al.*, 1998 Holland	66 ♀ 68 ♂	BMI tertiles	49±5 42±1	FFQ + 12 24-h recall BMRMeasured	—	—	BMI tertiles FFQ/UN 24hR/UN ♂ ♀ ♂ ♀ low 1.28 1.23 1.25 1.18 mid 1.22 1.08 1.15 1.07 high 1.11 1.07 1.13 1.03	BMI
Lindroos *et al.*, 1999 Sweden	9 ♀ 20 ♂	36±7 31±6	45±14 51±9	DH + UN 24-hr EE	-1	—	Prot 16 Fat 34 CHO 44	—
Little *et al.*, 1999 UK	58 ♀ 53 ♂ 61 CVDr 50 popul.	—	—	7-d FD BMREstimated	—	EI:BMR <1.2 All 40%/ Obe 60%/ No-obe 30%	—	BMI
Kretsch *et al.*, 1999 USA	22 ♀ Dif. race	11 Obe 34 11 N-Obe 21	30±8 26±4	7-d FD +7-d FD+ EB + EI observed	Obe -19±27% No-Obe -10±25%	—	—	Depression
Weber *et al.*, 2001 USA	16 ♀	8 Obe 32±4 8No-Ob 21±2	23±3 25±6	8-d FD+ DLW + body composition	After adjust for ΔEB: N-Ob NDS-30 N3-23% Obe NDS -38 N3 -39% NDS/Nut.data system N3-Nutritionist III	—	—	—
Johansson *et al.* 2001 Sweden	99 ♀ 94 ♂	~24.9±3.6 ~25.4±2.6	30-70	(10) 24h recalls BMREstimated URs EI:BMR<1.2	—	URs BMI<25 32% BMI 25-30 56% BMI >30 88%	—	Smoking BMI Age

Source/ Country	n	BMI (Kg/m2)	Age (y)	Methods	Energy Intake Misreporting (%)	Mis-reporters Prevalence (%)	Macronutrient Misreporting	Misreporting Determinants/ Comments
Martin *et al.*, 2003 Australia	31 ♀ 25 ♂ NIDDM	32±4 31±4	52±7 54±8	3-d FR + DH + BMREstimated	---	Urs DH FR No-obe 45 20% Obese 79 59%	---	URs prevalence increased over time by both methods
Caan *et al.*, 2004 USA	141 ♀ 302 ♂ Cancer prev. trials	—	>35	3-d FD BMREstimated	—	URs EI:BMR<1.06 ♀ ♂ Total 47 12% BMI<27 32 6% BMI>27 67 16%	—	—
Fricker *et al.*, 1989 France	896 ♀ 416 ♂	30±6 33±5	42±13 37±13	Diet history BMREstimated	0.0%	---	---	—
Male/female								
Lichtman *et al.*, 1992 USA	76 ♀ 14 ♂	10 diet-resist. 34±4 80 controls 36±7	48±12 47±12	14-d FD + DLW	Diet-resistant -47±16% Controls -19 ± 38%	---	---	---
Heitmann & Lissner, 1995 Denmark	171 ♀ 152 ♂	≈25 ≈25	50±11 50±11	DH ÷ UN + BMREstimated	Overall ≈23% ♂ -24 % ♀ -22%	Energy-UR's 85% Protein-UR's 72%	Protein ♂ +15 %E -14% (g) ♀ +8 %E -18% (g)	- % Body Fat
Adams, 1998 UK	85 ♂ 100 ♀ NIDDM	28 ± 5	45-79	Two 3-d FR BMREstimated	—	Urs ♂ 42% ♀ 59%	—	—
Tonstad *et al.*, 1999 Norway	205 ♀ 141 ♂ Lipid clinic	♀ 25±3 ♂ 26±3	54±8 46±10	4-d WR + BMREstimated	—	EI:BMR<1.5 ♀93% ♂91% EI:BMR<1.35 ♀88% ♂82%	—	Female sex BMI

Source/ Country	n	BMI (Kg/m2)	Age (y)	Methods	Energy Intake Misreporting (%)	Mis-reporters Prevalence (%)	Macronutrient Misreporting	Misreporting Determinants/ Comments
Tapsell *et al.*, 1999 Australia	27 ♀ 18 ♂	♀ 26.2 ♂ 27.8	46 47	3-d WR + Narrative DH + BMREstimated	---	Urs DH WR Baseline 20 11% 1 month 60 22% 2 months 40 24%	---	---
Kroke *et al.*, 1999 Germany	59 ♀ 75 ♂	26±5 27± 4	52± 5 56± 8	FFQ + 12(24-hr R) + DLW + UN	FFQ -20% 24-hR -26%	---	Prot (g): FFQ -21% 24-h recall -28%	—
Heitmann *et al.*, 2000 Denmark	65 ♀ 57 ♂	25±5 26±3	55±10 55±11	DH + UN BMREstimated	♀ -19.1% ♂ -22.2%	—	♀ -3.5 ♂ -5	---
Jonnalagadda *et al.*, 2000 USA	33 ♀ 45 ♂	17.3-39.8	22-67	3 multi-pass/ 24-hrR + EB	Self-selected diet ♀ -13% ♂ -11% Controlled-diet ♀+1% ♂-13%	—	—	—
Hise *et al.*, 2002 USA	32 ♀/22 ♂ Eating at cafeteria	30±3 30±3	22±4 23±4	14-d WR observer-recorded + DLW	♀ -3% ♂ +3 %	—	—	Prob. under-eating 62% of sample lost wt (no-significant.)
Female								
Lissner *et al.*, 1989 USA	63 ♀	---	19-67	3-5d FR+24-hr R Vs Observed	-23%	---	---	---
Buhl *et al.*, 1995 USA	10 ♀	33±7	39±12	14-d FD+DLW	-59±106%	100%	—	—

Source/ Country	n	BMI (Kg/m2)	Age (y)	Methods	Energy Intake Misreporting (%)	Mis-reporters Prevalence (%)	Macronutrient Misreporting	Misreporting Determinants/ Comments
Samaras *et al.*, 1999 UK	436 ♀	24±4	58±6	FFQ/7-d FR BMREstimated	BMRxPA-score -36% BMRx1.35 -39%	FFQ BMRxPAL normal weight 43% overweight 55% Obese 65% FFQ BMRx1.35 normal weight 22% Overweight 35% Obese 52% 7-dFR BMRx1.35: normal weight 18% Overweight 39% Obese 44%	7-dFR (N=197): URs VRs Fat 31.1±0.7 34.6±0.4% CHO 50.2±0.9 48.3±0.5% Prot 16.5±0.4 15.2±0.2%	BMI
Johnson *et al.*, 1998 USA	35 ♀ low-income	28±7	30±7	4 multi-pass/ 24-hr recall DLW	-16.9%	URs: 34% VRs: 63% ORs: 3%	—	- %Body Fat - Literacy
Taren *et al.*, 1999 USA	37 ♀ Dif. race	29±9	44±9	3-d FD DLW	- 11.6%	—	—	- Soc. desirability - Body image
Tran *et al.*, 2000, USA	35 ♀	28±7	30±7	4 multi-pass 24-hR (2 in-person/2-by tel.) +DLW	In person recall -18% Tel. Recalls -15%	—	—	—

Source/ Country	n	BMI (Kg/m2)	Age (y)	Methods	Energy Intake Misreporting (%)	Mis-reporters Prevalence (%)	Macronutrient Misreporting	Misreporting Determinants/ Comments
Caan *et al.*, 2000 USA	1137 ♀ Early stages of breast cancer	---	18-70	4 24-hr recall BMREstimated	---	Total sample 25.6% BMI <18.5 0.0% BMI 18.5-24.9 19% BMI 25-29.9 24% BMI 30-34.9 34% BMI 35-39.9 44% BMI ≥40 49%	---	Misreporting was more prevalent among: African-American Subjects weight fluctuations during adult life
Samuel-Hodge *et al.*, 2004, USA	200 ♀ NIDDM	35.7±8.0	59±10	3 24-hR Tel. administered TEEaccelerometer BMREstimated	---	URs 81% (EI:TEE<0.79) 58% (EI:BMR<SE-PAL) 92% (EI:BMR<1.27)	---	---
Male								
Goris *et al.*, 2000 Holland	30 ♂	34±4	44±7	7-d FD DLW EB	Under-reporting -37% Under-recording-12% Under-eating -26%	---	Fat Intake 39±6% no-underreporting 46%	---

♀= women; ♂=men; 7-d= seven-day; DLW= doubly labeled water method; BMR= basal metabolic rate; E= energy; RMR= resting metabolic rate; URs= underreporters; VRs= valid reporters; ORs=overreporters; BMI=body mass index; FFQ= food frequency questionnaire; EB= energy balance techniques; CHO= carbohydrate; 24-hR= twenty-four hour recall; UN= urinary nitrogen

Table 1.3 Studies in adult normal-weight subjects

Source	N	BMI (Kg/m2)	Age (y)	Methods	Energy Intake Misreporting (%)	Prevalence of Misreporters (%)	Macronutrients Misreporting (%)	Misreporting Determinants/ Comments
Male/female								
Schulz *et al.*, 1989 Germany	4 ♂ 2 ♀ Volunteer-paid	---	24 (20-30)	2 7-d WR+ DLW + 24-h HR	0.0 %	URs 33%/ ORs 50%/ VRs 17%	---	---
Livingstone *et al.*, 1990 UK	16 ♂ / 15 ♀	26±3 / 24±3	32±7 / 36 ±11	7-d WR + DLW	♂ -19% / ♀ -18%	URs: ♂38% ♀33% (ORs ♂ n=1)	---	---
Mertz *et al.*, 1991 USA	63 ♀ 203 ♂	---	21-64	7-d FR EB	♀ -18±16% ♂-18±19%	URs ORs VRs ♀ 75 / 11 / 14% ♂ 82 / 7 / 10%	---	---
De Vries *et al.*, 1994 Holland	150 ♀ 119 ♂	22±2 / 22±3	♂ 25±9 ♀ 26±10	3-d FWR ÷ EB	♀ -8±13% ♂-12±14 %	---	---	---
Seale & Rumpler, 1997 USA	10 ♀ / 8 ♂	23±3 / 26±1	52±5 50±7	7-d FR + DLW + 24-h Ind. calorimetry	EI-DLW ♀-11 ♂-12EI-E balan. ♀-11 ♂-12%	---	---	---
Black *et al.*, 1997 UK	18 ♀ 27 ♂	25±4 25±4	58±5 68±5	7-d WR + DLW + UN	♀ -11% ♂ -12%	EI Urs ♀ 39 ♂ 30% Protein URs♀33 ♂19%	Protein: ♀ -10 ♂ -15	---
Voss *et al.*, 1998a Germany	14 ♀ 14 ♂	---	56±8 52±5	FFQ + 24-hR + DLW	♀ -19.7% ♂ -20%	---	---	---

Source	N	BMI (Kg/m2)	Age (y)	Methods	Energy Intake Misreporting (%)	Prevalence of Misreporters (%)	Macronutrients Misreporting (%)	Misreporting Determinants/ Comments
Asbeck *et al.*, 2001 Germany	55 ♀ 28 ♂	22±2 / 24±2	26±3 27±3	7-d FR + DLW BMREstimated	♀ -11±22 ♂ -3±15% (♀URs -33 ♂URs –23)	URs ♀ 49 ♂ 14%	---	Restraint Disinhibition
Barnard *et al.*, 2002 Australia	8 ♀ / 7 ♂	19-33	22-59	7-d FR + DH + DLW	Total ♀ ♂ DH -23 -47 -1% FR -29 -41 -18%	—	—	High EE
Female								
Bingham *et al.*, 1995 UK	160 ♀ Presented in quintiles of UN/DN	All quintiles 24 ±1	50-65	Four 4-dWR+ UN + Dutch EBQ	EI/BMR 1 1.54±0.03 2 1.60±0.05 3 1.40±0.03 4 1.32±0.04 5 1.15±0.04	---	UN/DN 1 0.76±0.005 2 0.83±0.003 3 0.89±0.003 4 0.96±0.004 5 1.13±0.022	—
Ortega *et al.*, 1996 Spain	126 ♀	Tertiles <20.1 (n=38) 20.1-21.4(n=38) ≥21.4 (n=50)	19-36	7-dFR + BMREstimated	BMI tertiles Wt. conscious No-con Low -3±17 -9±16% mid -11±13 -5±13% high -16±20 -4±4%	—	—	—
Martin *et al.*, 1996 Canada	29 ♀	23±3	49±5	7-d FD + DLW	-21±18%	34.5%	—	—
Sawaya *et al.*, 1996 USA	10 ♀	21±2	25±4	FFQ + 7-d WR+ 24-h R + DLW	WR 24-h R FFQ -19 -13 -20%	—	—	—

Source	N	BMI (Kg/m2)	Age (y)	Methods	Energy Intake Misreporting (%)	Prevalence of Misreporters (%)	Macronutrients Misreporting (%)	Misreporting Determinants/ Comments
Black *et al.*, 2000 UK	48 ♀	24±3	50-65	Four 4-dWR + DH +DLW + UN + BMREstimated	(DLW n=16) WR -11 DH -2% PAL 1.65 WR: -12 DH: -11%	---	WFR: -6%N DH: +3%N	Observers in DH
Frost Andersen *et al.*, 2003 Norway	17 ♀	22±2	24±3	FFQ DLW	-10%	URs 47 / ORs 12 / VRs 41	---	---
Scagliusi *et al.*, 2003 Brazil	35 ♀		23-53	7-d FR 24-h Heart rate	-21%	URs 49%	---	Social desirability Under-eating
Lof & Forsum 2004 Sweden	37 ♀	23±3	21-41	(3) 24h recall DLW	--	URs (EI:EE=0.76) 48.6%	--	Constant under-reporting
Female: restrained								
Tuschl *et al.*, 1990 Germany	23 ♀ Unrestrained Restrained	21±1 / 20±1	25±4 22±1	3-d FWR + DLW + 3-FEQ	Restrained ≈ +5% Unrestrained ≈ +2%	---	---	---
Bathalon *et al.*, 2000 USA	60 ♀ unrestrained restrained	24±1 / 25±1	60±1 59±1	FFQ + 7-d WR + 24-h recall DLW	**EI-TEE** Unrest. /Restrain 7-dWR -11 -19% 24-hR -18 -24% FFQ -23 -26% **TEE+ΔBW** 7-dWR +7 -6% 24-hR -2 -13% FFQ -8 -14%	---	---	Low hunger Restraint

Source	N	BMI (Kg/m2)	Age (y)	Methods	Energy Intake Misreporting (%)	Prevalence of Misreporters (%)	Macronutrients Misreporting (%)	Misreporting Determinants/ Comments
Dieticians								
Goris & Westerterp, 1999 Holland	24 ♀ (dieticians)	22±2 (17-26)	34±9 (22-60)	7-d FR + BMRmeasured + PA by tracmor	UR -16±29% 100% Explained by undereating	---	---	---
Goris & Westerterp, 2000 Holland	18 ♀ (dieticians) prev. partic.	22±2 (17-26)	34±9 (22-60)	7-d FR BMRm PA by tracmor	No Underreporting EI/BMR= 1.56	---	---	---
Champagne *et al.*, 2002 USA	20 ♀ 10 dieticians 10 no-dietic.	23±1 / 23±1	36±4 33±2	7-d FR + DLW	Dieticians -10.4 % No-dieticians -18.5%	---	---	---
Athletes								
Edwards *et al.*, 1993 USA	9 ♀ Dist. runners	19±2	---	7-d FR DLW	-31.9%	---	---	---
Beidleman *et al.*, 1995 USA	20 ♀ dist-runners controls	22 22	22±1 24±1	3-d FR + TEM+ TEA+24-h HR+ BMRmeasured	dist-runners -33.6% controls -17.7%	---	---	
Hill & Davies, 1999 Australia	12 ♀ Ballet/danc.	22±2 (17-26)	---	4-d WR DLW	-21%	---	---	---
Hill & Davies, 2002 Australia	7 ♀ Elite rowers	~21	20±1	4-d WR DLW	Adjusted for EB -34%	URs 86%	---	---

Source	N	BMI (Kg/m2)	Age (y)	Methods	Energy Intake Misreporting (%)	Prevalence of Misreporters (%)	Macronutrients Misreporting (%)	Misreporting Determinants/ Comments
Post-obese female/male								
Black *et al.*, 1995 UK	10 ♀, 1 ♂	24±3 (19-28)	35±9	21-d WR + DLW	-27±17% (-53 to -5)	54.5% URs	UN/DN 1.15±0.42	---
Small/large eaters								
Clark *et al.*, 1994 Australia	(12 ♀) 6 small 6 large/eater	37±4 40±2	19 23	5-d WR ÷ DLW	Large-eaters +24% Small-eaters -46%	---	---	---

♀= women; ♂=men; 7-d WR= seven-day weighed record; DLW= doubly labelled water method; 3-d WR three-day weighed record; 24-hR= twenty-four hour recall; 24-h HR=twenty-four hour heart rate; URs= under-reporters; VRs= valid-reporters; ORs=over-reporters; EB= energy balance techniques; BMRe= basal metabolic rate estimated; BMRm= basal metabolic rate measured; DEXA= dual energy x-ray absorptiometry; CHO= carbohydrate; UR=underreporting TEM=Thermic effect of meals; TEA=Thermic effect of activity

Table 1.4 Older subjects Studies

Source	N	BMI (kg/m^2)	Age (y)	Methods	Energy intake Misreporting (%)	Prevalence of Misreporters %	Macronutrients Misreporting %	Misreporting Determinants/ Comments
Dietary methods and overall								
Pannemans & Westerterp, 1993 Netherlands	11 ♀ 17 ♂	26±3 25±3	70±5	4-dFR + FFQ + DLW + EB (faeces & urine)	FR -10 % FFQ -9%	—	—	—
Rothenberg *et al.*, 1998 Sweden	12 ♀ 8 ♂	25±3 25±3	73y	DH + DLW +24-h HT	-12%	VRs 58/URs 33/ORs 8%	—	—
Female/Male comparisons								
Goran & Poehlman, 1992 USA	6 ♀ 7 ♂	25±3 24±3	66±5	3-d FD +DLW +RMRm	♀-31±18 ♂-12±11%	—	—	—
Johnston *et al.*, 1994 USA	56 ♀ 81 ♂	25±3 24±3	66±6	3-d FR +RMRm +TEEe	♂ -11.7 ♀ -23.6%	—	—	% Body Fat
Rothenberg, 1994 Sweden	42 ♀ 34 ♂	—	70 y	FFQ + 4-d FR +4-d UN BMRe	All ♀ ♂ FFQ +10 +7 +17 % FR -14 -14 -12%	—	Prot. All ♀ ♂ FFQ +25 +15 +37% FR +7 +5 +9%	—
Tomoyasu *et al.*, 1999 USA	43 ♀ 39 ♂	25±1 25±1	69±1	3-d FR + DLW	♀-18±3 ♂-23±3%	URs ♀93 ♂83 % ORs (♀♂) 13%	—	BMI/ Fat mass Marital status
Tomoyasu *et al.*, 2000 USA	36 ♀ 28 ♂ Afr-Am	21-45	65±8	3-d + DLW + BMRm	♀ -10±30 ♂-14±31%	—	—	Low diet. fat High diet. protein
Luhmann *et al.* 2001 Germany	238 ♀ 105 ♂	24±1	60-89	3-d FR BMRestimated	—	♀ 7.6 % ♂ 16.2 %	—	Low education BMI

Source	N	BMI (kg/m^2)	Age (y)	Methods	Energy intake Misreporting (%)	Prevalence of Misreporters %	Macronutrients Misreporting %	Misreporting Determinants/ Comments
Seale *et al.*, 2002 USA	13 ♀ 14 ♂ Rural	28±3 28±2	74±4	3-d +DLW + BMRm	♀ -24±10 ♂-30±18%	---	---	---
Female								
Reilly *et al.*, 1993 UK	11 ♀	---	73±3	3-d WR+ DLW +BMRm	-26±17%	---	---	---
Kaczkowski *et al.*, 2000 Canada	53 ♀	24±4	65±11	4-d FRm-m+DLW	-24±23%	---	---	---
Visser *et al.*, 1995 Holland	12 ♀	26±5	74±3	DH + 24-h EE 2-d + UN + PA-diary	-12 ± 7%	---	NI/UN -13 %	---
Sawaya *et al.*, 1996 USA	10 ♀	24±3	74±4	FFQ + 7-d WR + 24-hR + DLW	WR -22%/ FFQ -2% / 24-hR -25%	---	---	Age

♀= women; ♂=men; 3-d FR= three-day food record; FRm-m=food record multi-media; RMRm= resting metabolic rate measured; DLW= doubly labeled water method; 3-d WR= three-day weighed record; BMRm= basal metabolic rate measured; FFQ= food frequency questionnaire; UN= urinary nitrogen; UN/DN=urinary nitrogen/dietary nitrogen; DN/UN= dietary nitrogen/urinary nitrogen; 24-h EE= twenty-four hours energy expenditure; PA= physical activity; BMI=body mass index; EI= energy intake; TEE= total energy expendirture; CHO= carbohydrate; URs= under-reporters; VRs= valid-reporters; ORs=over-reporters;

Table 1.5 Population studies

Source	Sample	BMI (kg/m2)	Age (y)	Methods	Energy Intake Misreporting	Prevalence of Misreporters %	Macronutrients Misreporting	Misreporting Determinants/ Comments
Non-obese/ Obese								
Stallone *et al.*, 1997 UK	406 ♀ 459 ♂ Whitehall II (11% N-white)	♀ 25±5 ♂ 25±3	♂50±6 ♀50±6	7-d FR + BMRestimated UR= EI/BMRx1.2	Overall 1.34 ± 0.33 Without UR's: 1.51 ± 0.26	All 33 / ♀35 / ♂32 BMI<20 ♀19 / ♂14% 20-24.9 ♀26 / ♂24% 25-29.9 ♀47 / ♂41% ≥30 ♀51 / ♂45%	---	Relative weight Socio-economic status
Kiesges *et al.*, 1995 USA	11663 ♂ & ♀ Racially mixed 4530 ♀	URs 27±6 VRs 24±4	50±17 43±18	24-hR + BMRestimated UR= EI/BMR<0.92	—	All 31 / ♀ 65 / ♂35%	—	—
Ballard-Barbash *et al.*, 1996 USA	1854 ♀	24	19-50	6 24-hR trough the year (1985-86) + BMRestimated UR=EI/BMR<1.06	---	All 52% Under-weight 29% Normal-weight 48% Over-weight 71%	---	---
Pomerleau *et al.*, 1999 Canada	15662 ♀ 14586 ♂	---	---	FFQ + BMRestimated URs EI:BMR<1.2	---	♀ 38% ♂ 43% BMI>25 34% 25-29.9 44% ≥30 51%	---	Age Nationality Urban/rural resid. Income

Source	Sample	BMI (kg/m2)	Age (y)	Methods	Energy Intake Misreporting	Prevalence of Misreporters %	Macronutrients Misreporting	Misreporting Determinants/ Comments
Subar *et al.*, 2003 USA	223 ♀ 261 ♂	---	40-69	24-h R multi-pass ÷ FFQ + DLW + UN	24-hR FFQ ♀ -16-20 -30-38% ♂ -12-14 -31-36%	BMI URs ♀ ♂ 18.5-24.9 13 13% 25-29.9 22 17% ≥30 35 33% 24-hR ♀ 22 ♂21 FFQ ♀49 ♂50 Protein URs 24-hR ♀10 ♂ 13 FFQ ♀29 ♂39	Protein intake 24-h recall FFQ ♀ 11-15 27-32 ♂ 11-12 30-34	BMI
Bedard *et al.*, 2004 Canada	140 ♀ 106 ♂	♀~23.6 / ♂ 26.8	18-82	FFQ + BMRestimated	--	BMI URs ♀ ♂ <25 30 40% 25-30 32 67% >30 73 65%	---	Age BMI Low education
Female/ male Heywood *et al.*, 1993 Australia	2356 ♂ 2813 ♀	---	25-64	24-hR BMRestimated	---	URs EI/BMR<1.53 ♂ 12% ♀ 24%	---	---
Fogelholm et *al.*, 1996 Finland & Hirvonen *et al.*, 1997 Finland	Year 1982: 640 ♂/677 ♀ Year 1992: 664 ♂/765 ♀	1982Vs 1992 URs ♀ 26±5 / 26±5 ♂ 26±4 / 27±4 VR's ♀ 25±4/ 25±5 ♂ 26±3/ 26±4	25-64	3-d FR + MET-values + BMRestimated UR= EI/BMR<1.28	---	URs (82 Vs 92) ♀ 34 / 47 ♂ 26 / 42% BMI<25 ♀ 23 / 38 ♂ 13 / 27% BMI>27 ♀ 50 / 58 ♂ 40 / 54%	% diet fat (82 Vs 92) ♀ 37 / 34 ♂ 39 / 34 excluding URs ♀ 38 / 35 ♂ 39 / 35	---

Source	Sample	BMI (kg/m2)	Age (y)	Methods	Energy Intake Misreporting	Prevalence of Misreporters %	Macronutrients Misreporting	Misreporting Determinants/ Comments
Briefel *et al.*, 1997 USA	3813 ♂ 3956 ♀ NHANES III Ethnic. mixed	---	≥ 20	24-hR (repeated in 623 subjects) BMRestimated UR=EI/BMR<0.9	---	URs ♂ 18% ♀ 28% (♂55% ♀58% URs were persistent i.e. under-reporting in 1st and 2nd 24-hR)	---	Age Education Socio-econ. status Ethnicity Body size Dieting
Price *et al.*, 1997 UK	938 ♀ 960 ♂	---	36-43	7-d FR + BMRestimated UR=EI/BMR <1.1	—	All 21% / ♀23% / ♂18 %	---	Low education Soc/class of origin
Lafay *et al.*, 1997 North France	501 ♀ 529 ♂	22±4 24±3 Self-assessed	♀34±8 ♂35±9	3-d FR + BMRestimated	---	UR=EI/BMR <1.05 ♀ 15.8% ♂ 16.3%	---	---
Pryer *et al.*, 1997 UK	1076 ♂ 1102 ♀	---	16-74	7-d FR BMRestimated UUN	♀ EI:BMR1.22 ♂ EI:BMR 1.39	♀ 46% ♂ 29%	UN/DN URs VRs ♀ 0.91±0.3 0.77±0.3 ♂ 0.92±0.5 0.72±0.2	---
Rothenberg *et al.*, 1997 Sweden	440 ♀ / 369 ♂ Studied in 1971, 1981, 1993 regarded as one sample	---	70y	DH BMRestimated	---	URsEI/BMR ♀ ♂ <1.52 57 56% <1.2 28 22% ORs ♀ >1.82= 8 % ♂ >2.1= 18%	—	---
Johansson *et al.*, 1998 Norway	1461 ♂ 1559 ♀	23±4 25±3	♀42±17 ♂43±16	FFQ + BMRestimated EI/BMR UR<1.34 ORs >2.4	♀ EI:BMR 1.58 ♂ EI:BMR 1.48	URs ORs ♂ 38 % 7% ♀ 45% 5%	—	BMI Age Wt change Desire Smoking

Source	Sample	BMI (kg/m2)	Age (y)	Methods	Energy Intake Misreporting	Prevalence of Misreporters %	Macronutrients Misreporting	Misreporting Determinants/ Comments
Voss *et al.*, 1998b Germany	2356 ♂ 2862 ♀	♂26±3 ♀25±4	♂53±8 ♀50±9	FFQ + BMRestimated	---	EI/BMR<1.35 ≈40%	---	---
Becker *et al.*, 1999 Sweden & Ireland	1651 Swedish 2212 Irish ♂ & ♀	---	15-74 47/48	7-d FR + FFQ + BMRestimated	---	EI URs EI/BMR Sweden <1.1 = 18% <1.34 = 53% Ireland <1.27 = 53%	---	---
Krebs-Smith *et al.*, 2000 USA	8334 ♂ & ♀	---	---	2 24-hR + BMRestimated EI/BMR UR<0.8	---	15%	---	Gender Age Education
Mennen *et al.*, 2000 UK	2885 ♂ & ♀ Rural/Cameroon Urban/Cameroon Jamaican UK-Afrocaribcan	---	20-74	FFQ BMRestimated	---	URs EI/BMR<1.15 RC UC J UK-AC ♀ 6 4 24 28% ♂ 6 5 19 39% EI/BMR<1.32 ♀ 8 7 34 46% ♂ 9 9 30 53%	---	BMI
Harrison *et al.*, 2000 Egypt/USA	♀ 4586 Egyptian 3010 American	---	18-60	24-h recall BMRestimated URs EI/BMR<0.92	---	URs Am Egypt BMI<25 25 7% 25-<30 35 9% >30 54 14%	---	BMI
Becker & Welten, 2001 Sweden/ Netherlands	300 ♂ & ♀	---	20-79	3-d FR BMRestimated	---	URs EI:BMR<1.04 11%	---	---

Source	Sample	BMI (kg/m2)	Age (y)	Methods	Energy Intake Misreporting	Prevalence of Misreporters %	Macronutrients Misreporting	Misreporting Determinants/ Comments
Luhrman *et al.*, 2001 Denmark	105 ♂ 238 ♀	27 ± 4	60-89	3-d FR BMRestimated EI/BMR UR<1.073	---	URs ♂16% ♀8%	---	Low-education BMI, Body fat Weight loss desire
McGowan *et al.*, 2001 North-South Ireland	1379 ♂ & ♀	---	18-64	7-d FR BMRestimated EI/BMR UR<1.1	~10	All 25 / ♀29 / ♂20% No dieter/ill All 21%	---	---
Hoidrup *et al.*, 2002 Denmark	175 ♂ 173 ♀	♂25±3 ♀24 ± 3	♂ 47±11 ♀ 56±11	7-d FR + DH TEEestimated	FR ♂ -16 ♀ -19 % DH ♂ -18 ♀ -22%	—	—	Age BMI
Ferrari *et al.*, 2002 Europe (EPIC)	35955 ♂ & ♀	---	35-74	24-h R + BMRestimated	---	URs ♀ 14% ♂10%	—	BMI Age
Vyas *et al.*, 2002 UK	239 ♀/169 ♂ origin European Afro-Caribbean Pakistani	♀ ♂ 30 27 27 29 27 30	25-79	FFQ BMRestimated URs EI:BMR<1.2	—	URs ♀ ♂ European 78 64% Afro-Car. 61 77% Pakistani 79 79%	—	---
Mendez *et al.*, 2004 Jamaica	359 ♀ 351 ♂	—	25-75	FFQ + BMRestimated URs EI/BMR<1.34 ORs >2.4	—	URs ORs VRs ♂ 23 24 54% ♀ 39 16 46%	---	—
Female								
Hulten *et al.*, 1990 Sweden	154 ♀ (1968/69) 205 ♀ (1974/75) 331 ♀ (1980/81)	---	---	DH (improved 1968-81) 24-h UN	EI (Kcals): (68-69) 2030±500 (74-75) 2150±505 (80-81) 2350±550	—	Prot 1968/1974/1980 BMI≤24 0/ +13/ +26 24-30 -10/ +3/ +17 >30 -22/ -6/ +5	---

Source	Sample	BMI (kg/m2)	Age (y)	Methods	Energy Intake Misreporting	Prevalence of Misreporters %	Macronutrients Misreporting	Misreporting Determinants/ Comments
Okubo & Sasaki 2004 Japan	1889 ♀	21±3	18-20	DH BMRestimated URs EI:BMR <1.27 ORs EI:BMR >2.4	--	URs 37% ORs 2.4%	--	BMI Body weight
Male								
Satia-Abouta *et al.*, 2002 USA	15266 ♂	---	55-79	FFQ + BMRestimated	---	URs 56.6%	---	Age, BMI, Phys. Act., Education,
Rosell *et al.*, 2003 Sweden	301 ♂	26 ± 3	63±1	7-d FR + BMRestimated	---	URs 29%	---	BMI History of dieting
Children								
Maillard *et al.*, 2000 France	221 ♀ 280 ♂	15.4 15.4	5-11	24-h recall BMRestimated	---	EI:BMR <1.5 ♀ 20% ♂ 17%	---	---
Matthys *et al.*, 2003 Belgium	212 ♀ 129 ♂	♀ ~21.5 ♂ ~20	13-18	7-d FR BMRestimated	—	EI:BMR <1.1 ♀ 20% ♂ 8% <1.35 ♀ 47% ♂ 19%	—	—
Elderly								
Cook *et al.*, 2000 UK	539 ♀ 558 ♂	—	≥65	7-d FR BMRestimated	—	EI:BMR <1.1 ♀ 48 ♂ 29% <1.2 ♀ 61 ♂ 41%	—	Obesity Social class

Source	Sample	BMI (kg/m2)	Age (y)	Methods	Energy Intake Misreporting	Prevalence of Misreporters %	Macronutrients Misreporting	Misreporting Determinants/ Comments
Pregnant women								
Winkvist *et al.* 2002 Sweden-Indonesia	490♀ Pregnant	Pre-pregnant 21 ± 3	29 ± 5	Six 24-h recall BMRestimated	---	URs EI:BMR <1.55 By trimester 1st 30%/ 2nd 16%/ 3rd 18%	---	---
Dietary Methods comparison								
MacIntyre *et al.*, 2003 South Africa	74 ♀&♂	---	15-65	FFQ + 7-d WR + BMRestimated	---	EI:BMR<1.2 FFQ 43% WR 28%	---	---

♀= women; ♂=men; DH=dietary history; ; UN= urinary nitrogen; EI= energy intake; BMI=body mass index; UR= underreporting; BMRe= basal metabolic rate estimated; URs= underreporters; LERs= low energy reporters; 24-hR= twenty-four hour recall; VRs= valid reporters; CHO= carbohydrate; Alc= alcohol; UUN= urinary urea nitrogen; UN/DN= urinary nitrogen/dietary nitrogen; SES= socioeconomic status

Chapter 2

Intentional mis-reporting of food consumption and its relationship with body mass index and psychological scores in women

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2.0 Context of the present chapter

This study was designed to answer in part the research questions in section 1.4. The other aspects of the research questions will be addressed later in this thesis.

Research questions for the present chapter

1. Do (some) people intentionally provide inaccurate or misleading information about food intake?

2. Does a declared inclination to over-report or to under-report relate to BMI, gender, age or psychological variables?

ABSTRACT

Background: The reasons for mis-reporting food consumption warrant investigation.

Objective: To document intention to mis-report food consumption and its associations with psychological measures in women.

Design: A total of 184 female volunteers aged 18-65 y, comprising 50 seeking help in primary care to lose weight with a body mass index (BMI) ≥ 30 kg/m^2 (obese-clinical group) and 134 nurses (non-clinical groups) (BMI < 25 kg/m^2, n = 52; BMI 25-29.9 kg/m^2, n = 45; BMI ≥ 30 kg/m^2, n = 37) were studied. A questionnaire was administered containing 3 psychological tests (self-esteem, psychological well-being and Stunkard's three-factor eating questionnaire) and new items to address food intake mis-reporting.

Results: Overall, 68% of participants declared an inclination to mis-report (64% non-clinical, 78% clinical). Inclination to under-report was 29%, 33% and 51% in the three non-clinical groups; and 46% among the obese clinical patients. Among the same groups inclination to over-report were 39%, 29%, 11%, and 32%. After adjusting for social deprivation and BMI, women inclined to mis-report had higher hunger (P=0.008) and disinhibition (P=0.005) scores than those intending to report accurately. These variables were associated with current dieting, frequency of dieting, self-reported bingeing and dissatisfaction with body weight.

Conclusions: These findings indicate that intentional under-reporting of food consumption was most common among obese women while over-reporting was most common among thinner individuals; both were associated with eating behaviour. Current dieting, frequency of dieting in the past, self-reported bingeing and dissatisfaction with body weight seem to mediate this relationship.

2.1 INTRODUCTION

An apparent inverse relationship between body mass index and food consumption has been a common finding in diet surveys (Keen *et al.*, 1979; Romieu *et al.*,1988). This pattern is still being reported despite a huge increase in obesity prevalence (Prentice & Jebb, 1995). Substantial dietary under-reporting or temporary under-eating during surveys is now a well-recognised contributory problem to these patterns (Macdiarmid & Blundell, 1998). Consistent dietary mis-reporting affects the interpretation of the relationship between nutrition parameters and the aetiologies of many chronic diseases. For example, the increasing prevalence of obesity casts uncertainty on the interpretation of data, which suggests steadily falling food consumption in the UK (Prentice & Jebb, 1995). The issue is even more complicated if there is selective mis-reporting of specific nutrients, e.g. fat (Willett, 1997; Willett, 1998; Bray & Popkin, 1999).

Figures for dietary mis-reporting vary across studies depending on the criteria used. Commonly, an arbitrary cut-off is chosen for the ratio of energy intake (EI) to basal metabolic rate (EI/BMR). Recent surveys report high rates of dietary mis-reporting in the general population. For example, 38% of under-reporting (EI/BMR < 1.35) in men and 45% in women, while over-reporting (EI/BMR > 2.4) is less frequent, only 7% of men and 5% of women (Johansson *et al.*, 1998). However, the prevalence of under-reporters varies among some subgroups of the population, particularly more so in the obese (Macdiarmid & Blundell, 1998).

Few studies have explored the reasons for dietary mis-reporting. Decreased habitual food consumption during the recording period (i.e. under-eating) has been reported as more prevalent than failure to record in the food diaries (under-recording) in lean women (Goris & Westerterp, 1999) and obese men (Goris *et al.*, 2000). Embarrassment

and the nuisance of recording food intake are among the reasons given by both male and female under-reporters (Macdiarmid & Blundell, 1997). Recently, social desirability bias, the tendency of individuals to distort responses to comply with societal norms in order to avoid criticism, has been associated with under-reporting in women (Taren *et al.*, 1999). Based on this evidence, Macdiarmid and Blundell (1998) have proposed the classification of dietary mis-reporting as intentional or non-intentional.

The current study investigated the intention to mis-report food consumption, and its association with measures of self-esteem, psychological well-being (depression, anxiety, positive well-being) and eating behaviour (dietary restraint, disinhibition and hunger) in women with a range of BMI not seeking weight management. Comparisons were made with a group of obese women currently attending weight management groups.

2.2 Materials and Methods

2.2.1 Subjects

Convenience samples of female nurses (non-clinical groups) and obese women seeking help for weight loss (clinical group) were recruited for this study. Participants were approached in person and recruited consecutively after voluntarily accepting an invitation to answer a questionnaire on eating habits applied in a one to one interview. Participants in the non-clinical group were recruited at their workplace at a General Hospital, while the clinical group was recruited from primary care centres, all in Glasgow, Scotland. In total, 184 participants aged 18-65 years were included. The clinical group comprised 50 women with a BMI $\geq$ 30 (obese) attending weight management groups. No other inclusion criteria were established for these subjects. The non-clinical group comprised 134 participants, 52 with a BMI 18.5-24.9 (normal

weight), 45 with BMI 25-29.9 (overweight), and 37 with BMI ≥ 30 kg/m^2 (obese) not enrolled in, or seeking, weight management or following a special diet.

The response rate was higher among the clinical (80%) than among the non-clinical group (50%). The reasons given for non-participation were mainly related to embarrassment of being weighed, with most of the non-clinical women who rejected participation being apparently overweight or obese.

The recruitment method did not attempt to ensure representative samples of the groups under study, because this was not considered strictly necessary in order to test hypotheses relating BMI to mis-reporting, and because experience showed low response rates with randomised and self-completed questionnaires on this topic. Instead, the method chosen aimed to achieve high response rates by a personal approach and specifically to ensure a wide and balanced range of BMI within the non-clinical group, which could not be achieved by postal approaches.

The Glasgow Royal Infirmary Research Ethics Committee approved this study. After describing the procedures, signed consent was obtained from all participants.

Social deprivation or deprivation category (DEPCAT), a measure of affluence, was determined from postcode using an established method for Scotland (McLoone, 1997) with a scale ranging from 1 (highest affluence) to 7 (lowest affluence).

2.2.2 Questionnaire

Using an in-person interview approach, a questionnaire was applied on a single occasion. The questionnaires were applied in a one-to-one basis. It contained three well-established tests: the Rosenberg's Self-esteem Scale (RSS) (Rosenberg, 1965), the Psychological Well-Being Questionnaire (PWBQ) (Bradley & Gamsu, 1994), and the 3-

Factor Eating Questionnaire (3-FEQ) (Stunkard & Messick, 1985) **[Appendices 1-3]**; plus new items to determine intention to mis-report.

Briefly, the RSS provides a measure of global self-esteem. It is a 10-item self-report questionnaire containing multiple choice answers rated by 4-point Likert scales scored as 4 for "strongly-agree", 3 for "agree", 2 for "disagree" and 1 for "strongly-disagree". The range of possible scores for this scale was from 10 (indicating very low self-esteem) to 40 (maximal self-esteem).

The PWBQ is a 22-item scale that assesses psychological well-being. It was originally developed for type 2 diabetic patients but does not include questions related to diabetes (Bradley & Lewis, 1990). Thus it was considered suitable for wider use in the general population, and specifically in relation to obesity since eight out of the ten commonest symptoms of type-2 diabetes are related to BMI (UKPDS Group, 1990). The PWBQ has been translated into more than twenty languages and is considered to be sensitive to its sub-scales: depression (6 items), anxiety (6 items), positive well-being (6 items) and energy (4 items). These items are rated by a Likert scale from 0 (not at all) to 3 (all the time), with a range of possible total scores from 0 to 18 for depression, anxiety and positive well-being, from 0 to 12 for energy and from 0 to 66 for general well-being which is a single total score derived from the four sub-scales. Higher scores indicate a greater measure; the procedure for scoring this scale has been described by Bradley & Gamsu (1994).

The 3-FEQ, also known as the eating inventory, is a 51-item instrument to measure eating behaviour developed in lean and obese people. It includes three scales or factors with high reliabilities (derived from factor analysis); factor I indicating cognitive or dietary restraint (21 items), factor II representing disinhibition of control (16 items), and

factor III indicating perceived hunger (14 items). Thirty-six items require answers provided in a true-false basis whereas the other 15 employ a Likert scale for frequency. The range of possible scores ranges from 0 to 21 for restraint, from 0 to 16 for disinhibition, and from 0 to 14 for hunger, with higher scores indicating higher measures as described by Stunkard & Messick (1985). The two dietary restraint subscales, denoted as "flexible control" and "rigid control", proposed by Westenhoefer (1991) were also estimated.

An additional new item was included to assess inclination to mis-report food consumption using a multiple option answer:

If I ask you "how much food do you eat" would you be inclined to give an answer ____ than you probably do"?

A) a lot more B) a bit more C) a bit less D) a lot less

E) Other, specify_____

All the participants choosing the option "other" concurred in specifying the intention of reporting exactly what they eat. The option "accurate" was deliberately not included among the multiple answers to the above question with the intention to direct participants towards deciding whether they would over-report or under-report.

Body weight, recorded in kilograms, was measured using a Salter portable digital scale (Salter Ltd, Kent); waist circumference, in centimetres, was measured with an inelastic measuring tape; and height, recorded in metres, by using the Leicester portable stadiometer (Invicta Plastics Ltd, Leicester). All these anthropometric measurements were performed according to standard recommendations (WHO, 1995).

Information relating to the presence or absence of dieting at the time of the study, the number of previous attempts to lose weight, binge eating, dissatisfaction with body weight, and willingness to record food consumption data were recorded using the following questions:

"Are you currently on a diet to lose weight?" □Yes □No

"How many times have you tried to lose weight in the last two years?" (Open question)

"While eating, have you ever experienced losing control and you end up eating more than usual?" □Yes □No

"How satisfied are you with your current body weight?" (5-point Likert-scale ranging from "strongly satisfied" to "strongly dissatisfied").

If, right now, you were invited to participate in a study to record your food intake by weighing or measuring it for instance between 3 and 7 days, would you be willing to participate? □Yes □No If your answer is no, please explain your reasons

_____

Demographic information such as date of birth, marital status, and postcode was also obtained.

2.2.3 Statistical analysis

Normality tests (Shapiro-Wilk statistic) were performed before analysis. ANOVA analysis was initially carried out to compare the physical characteristics between the non-clinical groups while t-tests were used to compare the two obese groups. Chi-square was used for assessing relationship between categorical variables such as BMI (18.5-24.9, 25-29.9, ≥30) and the inclination to report categories (over-, under-,

accurate-report). Comparisons of psychological scores between groups based on their intention to mis-report were assessed using multivariate analysis of variance. Scores for psychological measures were entered as dependent variables and intention to mis-report as independent variable. Adjustments were made for age, social deprivation, and BMI, entering these variables into the analysis as covariates. A significance level of 0.05 was used in the ANOVA while for each significant psychological measure the groups were compared by examining the 95% confidence intervals for the group means. All statistical comparisons were carried out with SPSS 9.0 for windows (SPSS Inc., Chicago, Ill, USA).

2.3 Results

2.3.1 Physical characteristics

Results for the physical characteristics according to BMI category are shown in **Table 2.1**. Participants in the normal-weight group were a mean of 4, 6 and 13 years younger than those in the overweight non-clinical, obese non-clinical and obese clinical groups respectively. A similar pattern in social deprivation score across BMI categories was observed. In addition a significant difference in height was found between the non-clinical groups; post hoc analysis showed that the normal-weight group was significantly taller than the overweight group. Apart from age and social deprivation, no other significant differences were observed between obese clinical and non-clinical women.

2.3.2 Prevalence of intentional mis-reporting

Given the small number of respondents in the extreme categories, i.e. "a lot less" and "a lot more", of the question assessing intention to mis-report, participants were condensed

into 3 groups describing intention to over-report, accurate-report and under-report, respectively. The group "accurate" comprised participants selecting the option "other" and providing statements such as "I would report accurately", "I would tell the truth" or "I would report what I ate". **Figure 2.1** shows the prevalence of reporting intention by BMI in the clinical and non-clinical groups. A total of 68% of all participants reported an inclination to mis-report and only 32% (n=59) said they would report food intake accurately. A significant relationship between BMI category and inclination to mis-report was observed within the non-clinical groups (χ^2 = 10.2, P = 0.038), where intentional under-reporting, although common in all BMI categories, was significantly more prevalent among the obese non-clinical group compared to the normal and overweight groups. Prevalence of under-reporting was similar in obese women in the clinical (51%) and obese non-clinical group (46%), both figures higher than the normal-weight and overweight non-clinical groups.

A surprising finding was the high frequencies for participants reporting an inclination to over-report their food intake. This was most frequent in the non-clinical normal weight women; however a high proportion of the clinical group (obese) (32%) reported an intention to over-report too. Adjusting for age did not affect the relationship between BMI and the prevalence of inclination to mis-report.

2.3.3 Willingness to participate in dietary studies

Participants were asked whether they would agree to participate in a study recording food intake. **Table 2.2** shows that in the highest BMI category of the non-clinical group a lesser proportion of participants declaring an inclination to report food intake accurately, agreed to participate in a study recording their food intake. The reasons provided were mostly related to "lack of time" (60%, n=35), "cannot be bothered

recording food intake" (19%, n=11), "lack of interest" (16%, n=9), and "other" (5%, n=3). Overall, a high proportion of people (42%, n=25) who reported intention to report food accurately said they would not be willing to participate.

2.3.4 Psychological variables and intentional mis-reporting.

Based on the inclination to report food intake, scores for the psychological/behavioural questionnaires were compared by multivariate analysis of variance. Adjustments for age, social deprivation and BMI were made. **Table 2.3** shows that only differences in the disinhibition and hunger factors of the 3-FEQ were significant between groups. Comparisons between groups showed that compared to accurate-reporters, intentional under-reporters scored significantly higher for disinhibition, while over-reporters scored significantly higher for hunger.

2.3.5 Dieting, frequency of dieting, self-reported binge eating and dissatisfaction with current body weight

Table 2.4 shows the multivariate analysis of variance for hunger and disinhibition adjusting for several variables in addition to age, social deprivation and body mass index. Changes in the scores after adjustment for dieting, frequency of dieting, and bingeing indicate a partial association with disinhibition and hunger in this sample. Adjusting for dissatisfaction with body weight explained differences in disinhibition, while only adjusting for all of these variables explained differences in hunger.

2.4 Discussion

In many walks of life, people are reluctant to reveal details about personal behaviours. These include indulgent behaviours or value-laden personal activities but also possibly behaviours linked to personal safety and security-including food access, total food

consumption and specific foods. In the present study a substantial proportion of women, up to half the obese and one third of the lighter groups, declared an inclination to mis-report food consumption. These findings are comparable with previously reported intentional misreporting rates (Macdiarmid & Blundell, 1997). Our sample included a wide range of BMI as well as women recruited from clinical weight management groups; however a limitation was that these were not randomly selected. The methods employed direct contact to achieve a high response rate and ensure a wide range of BMI, which could not otherwise have been obtained by a postal approach. The questions employed were designed to direct participants towards deciding if they would be likely to under- or over-report, with accurate-reporting only declared after rejecting these options. Different results might have been obtained if the option "accurate" had been presented as a prompted response option or if the question had been asked in the context of actual food intake recording.

Macdiarmid & Blundell (1997) reported that after confronting participants with their results, 46% admitted mis-reporting food consumption. Other studies also provide indirect evidence of intentional mis-reporting. In a clinical study in obese subjects (Muhlheim *et al.*, 1998), EI was under-reported despite the use of a "bogus pipeline paradigm" (doubly labelled water technique), an experimental procedure leading the participants to believe that an experimenter could verify (pipeline) their responses and thus motivating them to report more accurately. Similar under-reporting rates (control vs experimental group) were found during the screening week. A significant between-phases difference was observed during the 2nd week in the experimental group (i.e. 45 vs 39%, P= 0.046); however based on their predicted intake participants were still under-reporting (P<0.0005). The clinical setting is one of the most difficult environments to assess food intake and women who attend slimming clinics often adopt

apparently extreme and untenable beliefs about their food consumption; a challenge to most doctors and dietitians working on dietary assessment and change of eating habits.

Deliberate under-eating during the recording period seems to be another class of intentional mis-reporting. In normal-weight female dietitians (Goris & Westerterp, 1999), under-eating accounted for total EI under-reporting. In a second study (Goris and Westerterp, 2000), after confronting these participants with their results no evidence of under-reporting was found. An apparent accurate recording of food led to the conclusion that under-reporting was not necessarily the result of a dishonest action; however factors such as social desirability or seeking of social approval might have been involved as shown earlier (Hebert *et al.* 1997).

Under-reporting may persist over time and by different dietary methods. A study in women throughout the four seasons of the year showed that 45% of the sample under-reported EI; 25% under-reported EI on the four occasions (Black & Cole, 2001).

2.4.1 Reasons for mis-reporting

Some studies have shown a relationship between under-reporting and psychological and/or behavioural measures. Macdiarmid & Blundell (1997) found that a higher score for dietary restraint was related to higher self-reported consciousness about eating habits. Dietary restraint has also been associated with underreporting in a French population study (Lafay *et al.*, 1997). Opposing these findings, Taren *et al.* (1999) found no association between dietary restraint and reporting accuracy; similarly Mela & Aaron (1997) reported no significant differences in the scores for anticipated honesty and accuracy in reporting food intake among restrained or unrestrained eaters, current or past dieters, overweight or not (BMI > 25 or ≤ 25). In addition, a positive correlation ($r=0.73$, $P=0.01$) between depression and EI mis-reporting in obese women has been

reported (Kretsch *et al.*, 1999). The current study found no relationship between restraint, depression and intention to mis-report.

The present study showed a relationship between intention to mis-report food consumption and the hunger and disinhibition factors of the 3-FEQ. Disinhibition has been associated with weight gain, while hunger was related to binge eating (Stunkard & Messick, 1985); with dysphoria (r =0.47, P=0.0001) and binge eating (r=0.70, P=0.0001), while hunger was associated with depression (r=0.32, P=0.0001) and binge eating (r = 0.46, P = 0.0001) (Foster *et al.*, 1998); with bulimia in women (Williamson *et al.*, 1995) and recently to EI under-reporting in non-obese men (Asbeck *et al.*, 2002). In the present study, disinhibition and hunger were related to variables such as dieting, frequency of dieting during the previous two years, self-reported bingeing and body weight dissatisfaction. Adjusting for the effects of all of these factors explained between-group differences in both, disinhibition and hunger scores.

Increases in anxiety, feelings of rebelliousness, defiance and desire to challenge the limitations set by their diet has been reported in restrained subjects following a pre-load leading to disinhibition (Ogden & Greville, 1993). Whether a dysphoric or a distressed status caused by disinhibition, can lead to participants to declare the possibility of mis-reporting food consumption is unknown. The possibility that declaring an accurate reporting might have provided a socially desirable answer in the present study cannot be resolved. In terms of social desirability or seeking of social approval, the findings of the present study are unusual and the high proportion of women declaring an intention to under- or over-report seems to be at odds with this theory. However it is possible that declaring an intention to mis-report is more socially acceptable than to give an accurate account of food consumption.

Recent studies provide evidence of an important role for disinhibition. In adult women disinhibition has been associated with greater weight gain and BMI (Hays *et al.*, 2002). Maternal dietary disinhibition and BMI have been found as strong predictors of daughters' overweight (Cutting *et al.*, 1999). In addition, disinhibition was strongly associated with obesity and was the most heritable factor of the 3-FEQ in the Amish religious community, indicating genetic components in eating behaviour (Steinle *et al.*, 2002).

In the present study an important proportion of participants inclined to mis-report said they would respond affirmatively to an invitation for a diet study reporting food consumption. This implies that participants inclined to mis-report are most likely to enrol in dietary surveys. The mixture of over- and under-reporters might not be apparent in mean results. Given the prevalence of mis-reporting and the identification of correlates such as restraint, social desirability, hunger and disinhibition, it seems that intentional reasons are more relevant than the non-intentional ones in the mis-reporting of food consumption. Non-intentional mis-reporting may still be significant in specific groups in which a decline in factors such as memory is a frequent result, e.g. the elderly.

The results of this study are important for practicing dietitians and policy makers. Perhaps the most important practical consequences of this study is to question the current practice for rejecting the dietary records of people who report food consumption below a threshold such as EI/BMR < 1.3 or 1.0. Such individuals are undoubtedly reporting food consumption less than required, on average, for energy balance and there may be selective mis-reporting for specific foods, food groups or nutrients. Our data suggest that this under-reporting is commonly intentional (at least in some population groups), but also that intentional under-reporting occurs commonly in those with

EI/BMR above 1.3. Furthermore, intentional over-reporting is common across all BMI categories- and particularly for those who are thinner or have lower BMR and apparently normal food consumption.

Future research needs to be directed towards identifying mis-reporters prospectively in diet studies and towards assessing the bias which may result from refusal to participate. It may be easier to obtain information about specific foods which people may mis-report rather than inclination to mis-report specific nutrients.

2.5 New research questions raised by this chapter

The study described in chapter 2 addressed the question of what is the significance of intentional dietary mis-reporting and its psychological and behavioural determinants. The high prevalence of intentional mis-reporting over non- intentional reasons is quite clear and supported by previous studies. The high levels of inclination to over-report food intake was unexpected. The possibility exists that some subgroups of the population may be less prone to intentionally or unintentionally mis-report. Some groups may have better skills or a better knowledge and therefore of nutrition.

Research questions:

1. Do men show different patterns of inclination to mis-report than women?

2. Do people with food related-occupations who might influence food choice of others exhibit different patterns of intentional dietary mis-reporting?

Table 2.1 Physical characteristics in female clinical and non-clinical groups (n=184) classified by BMI

	Non-clinical groups				Obese clinical group	
	BMI < 25	**BMI 25-29.9**	**BMI ≥ 30**		**BMI ≥ 30**	
	n = 52	**n = 45**	**n = 37**	**P [b]**	**n = 50**	**P [c]**
Age (y)	33.5 (9.7)	37.8 (9.1)	39.3 (8.9)	0.009	46.7 (12.5)	0.006
Deprivation Category [a]	4.3 (1.7)	4.6 (1.5)	5.2 (1.1)	0.02	6.1 (0.9)	0.001
Body weight (kg)	58.6 (6.4)	68.5 (6.7)	86.4 (13.6)	<0.001	87.7 (12.2)	0.641
Body height (m)	1.63 (0.07)	1.58 (0.06)	1.60 (0.06)	0.009	1.60 (0.06)	0.855
Body mass index (kg/m^2)	22.2 (2.1)	27.3 (1.1)	33.7 (4.9)	<0.001	34.1 (4.1)	0.809
Waist Circumference (cm)	70.6 (5.1)	84.8 (3.2)	94.0 (6.9)	<0.001	96.8 (8.1)	0.130

Values are means (s.d.). n = number of observations in each group

[a] Estimated from postcode, range 1 (highest level) to 7 (lowest level) (McLoone, 1997)

[b] Analysis of variance comparing only non-clinical groups

[c] T-test comparing only the clinical and non-clinical obese groups (BMI≥30)

Table 2.2 Answers [n (%)] to the question "Would you participate in a study recording food intake?"

	Non-clinical group						Clinical group		P-value
	BMI < 25		**BMI 25-29.9**		**BMI ≥ 30**		**BMI ≥ 30**		
	n = 52		**n = 45**		**n = 37**		**n = 50**		
	YES	NO	YES	NO	YES	NO	YES	NO	
All subjects	41 (79)	11 (21)	34 (76)	11 (24)	13 (35)	24 (65)	38 (76)	12 (24)	<0.001
Reporting intention									
More (Over-report) (n=53)	15 (75)	5 (25)	12 (92)	1 (7)	3 (75)	1 (25)	13 (81)	3 (19)	
Accurate report (n=59)	13 (77)	4 (23)	9 (53)	8 (47)	5 (36)	9 (64)	7 (64)	4 (36)	
Less (Under-report) (n=72)	13 (87)	2 (13)	13 (87)	2 (13)	5 (26)	14 (74)	18 (78)	5 (22)	

P-value for Chi-square test

Table 2.3 Psychological scores by inclination to mis-report, controlling for age, deprivation, and BMI (Analysis of variance)

	Likelihood to report:			
	More (Over-report)	**Less (Under-report)**	**Exact (Accurate-report)**	***P***
	N = 53	**n = 72**	**n = 59**	
Self-esteem	25.6 (25.0, 26.1)	25.8 (25.3, 26.3)	26.4 (25.9, 26.9)	0.052
3-Factor eating questionnaire				
Dietary restraint (Factor I)	8.9 (7.6, 10.2)	9.5 (8.4, 10.7)	8.9 (7.7, 10.1)	0.696
Flexible control	2.6 (2.1, 3.1)	3.1 (2.7, 3.5)	2.7 (2.3, 3.2)	0.340
Rigid control	3.0 (2.5, 3.6)	2.9 (2.5, 3.4)	2.8 (2.3, 3.2)	0.736
Disinhibition (Factor II)	7.6 (6.6, 8.5)	7.7 (6.8, 8.5)	5.8 (4.9, 6.7)	*0.007*
Hunger (Factor III)	6.0 (5.1, 7.0)	5.7 (4.9, 6.4)	4.0 (3.2, 4.9)	*0.004*
Psychological well-being questionnaire				
Depression	5.5 (4.7, 6.3)	5.0 (4.3, 5.7)	4.9 (4.2, 5.7)	0.532

	Likelihood to report:			
	More (Over-report)	**Less (Under-report)**	**Exact (Accurate-report)**	***P***
Anxiety	5.9 (4.9, 6.9)	5.4 (4.5, 6.2)	4.4 (3.4, 5.3)	0.077
Energy	6.1 (5.4, 6.8)	6.3 (5.7, 6.9)	7.2 (6.6, 7.8)	0.056
Positive well-being	11.0 (10.0, 12.0)	12.0 (11.2, 12.9)	12.1 (11.2, 13.1)	0.185
General well-being	41.8 (38.9, 44.6)	44.0 (41.5, 46.4)	46.1 (43.4, 48.8)	0.098

Values are mean (95 % confidence interval for group means)
Range of possible scores: self-esteem (10-40), dietary restraint (0-21), flexible/rigid control (0-7), disinhibition (0-16), hunger/ depression/ anxiety/ positive well-being (0-18), energy (0-12) and general well-being (0-66). Higher scores mean a higher measure.

Table 2.4 Disinhibition and hunger scores by inclination to misreport, controlling for different variables (Analysis of variance)

	Likelihood to report:			
	More (Over-report)	Less (Under-report)	Exact (Accurate-report)	*P*
	n = 53	n = 73	n = 59	
Current dieting[1]				
Disinhibition (Factor II)	7.5 (6.6, 8.5)	7.6 (6.8, 8.5)	5.9 (5.0, 6.8)	0.011
Hunger (Factor III)	6.0 (5.0, 6.9)	5.6 (4.8, 6.4)	4.1 (3.3, 5.0)	0.010
Frequency of dieting[1]				
Disinhibition (Factor II)	7.9 (7.0, 8.8)	7.1 (6.3, 7.9)	6.1 (5.3, 7.0)	0.021
Hunger (Factor III)	6.3 (5.3, 7.3)	5.4 (4.6, 6.3)	4.3 (3.4, 5.2)	0.011
Bingeing[1]				
Disinhibition (Factor II)	7.6 (6.7, 8.5)	7.4 (6.7, 8.2)	6.1 (5.3, 6.9)	0.022
Hunger (Factor III)	6.1 (5.1, 7.0)	5.5 (4.8, 6.3)	4.2 (3.3, 5.0)	0.008
Dissatisfaction with current body weight[1]				
Disinhibition (Factor II)	7.4 (6.5, 8.4)	7.5 (6.7, 8.3)	6.1 (5.3, 7.0)	0.053
Hunger (Factor III)	6.0 (5.0, 6.9)	5.6 (4.8, 6.4)	4.2 (3.3, 5.1)	0.015

	Likelihood to report:			
	More (Over-report)	Less (Under-report)	Exact (Accurate-report)	*P*
All variables[1,2]				
Disinhibition (Factor II)	7.8 (6.9, 8.6)	6.9 (6.2, 7.7)	6.5 (5.6, 7.3)	0.089
Hunger (Factor III)	6.1 (5.2, 7.1)	5.3 (4.5, 6.2)	4.6 (3.7, 5.5)	0.063

Values are mean (95 % confidence interval for group means)
[1] Control for each of these variables was carried out in addition to age, deprivation, and BMI
[2] Dieting + Frequency of dieting + bingeing + dissatisfaction with body weight

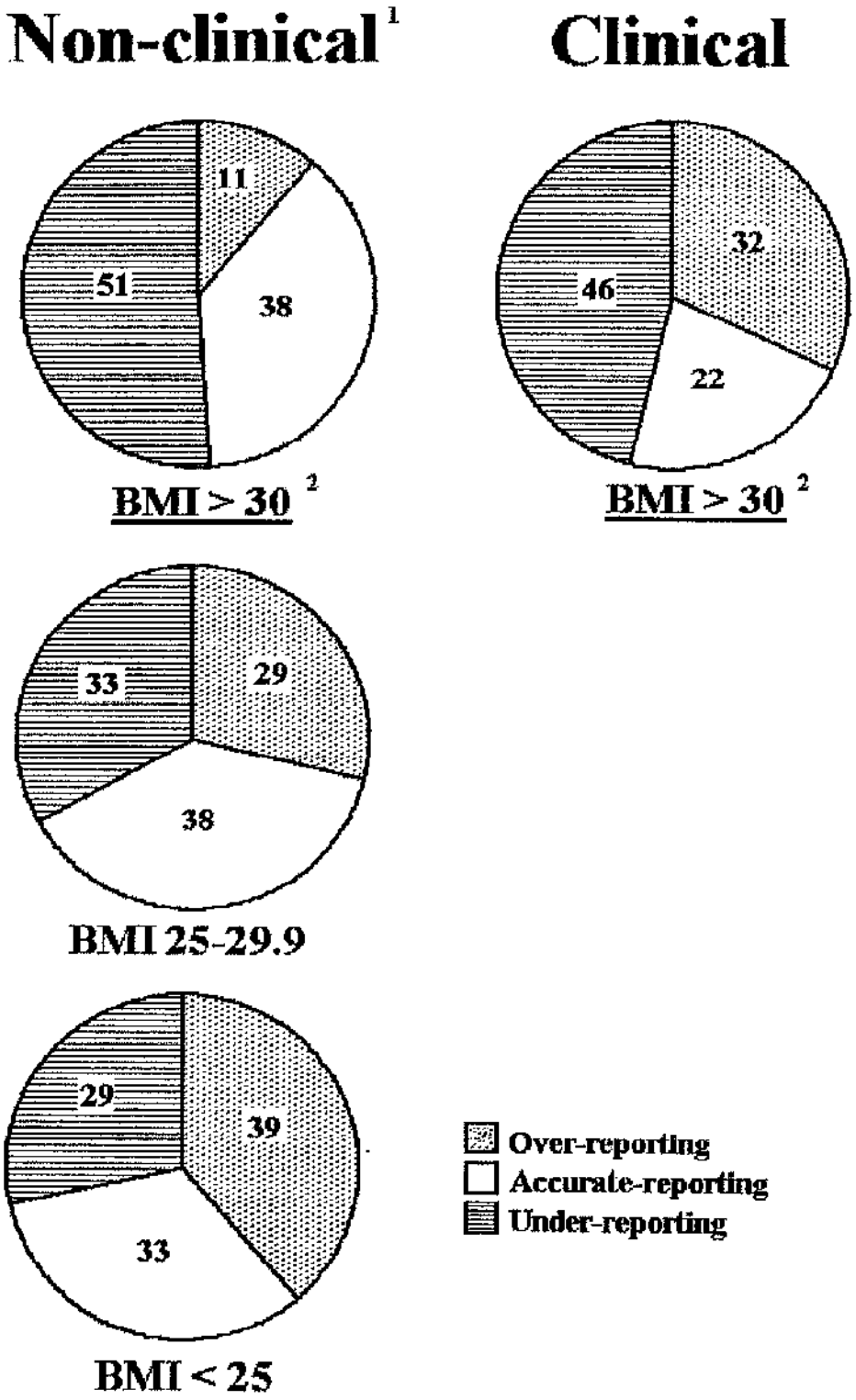


Figure 2.1 Prevalence (%) of intentional mis-reporting by body mass index (BMI) class.

[1] Non-clinical (nurses) BMI-groups $\chi^2 = 10.2$, P = 0.038
[2] Both obese (BMI ≥ 30) groups $\chi^2 = 6.135$, P = 0.047

Chapter 3

Dietary under-reporting in male subjects involved in food-related occupations

This work has not been published in a peer-reviewed journal, but it is presented here in the format for submission to Journal of Human nutrition and Dietetics

3.0 Context of this chapter

This study was designed to address the research questions in section 2.5

Research questions:

1. Do men show different patterns of inclination to mis-report than women?

2. Do people with food related-occupations who might influence food choice of others exhibit different patterns of intentional dietary mis-reporting?

ABSTRACT

Aim: To estimate dietary under-reporting in men involved in food-related occupations, whose dietary practices and capacity to influence the diet of others may be influenced by their own occupational expertise.

Methods: Fifty four subjects, 18 butchers, 18 fishmongers and 18 schoolteachers (age 39-53 years, BMI 23.2-27.4 kg/m^2) completed a FFQ and a 7-d food diary. Under-reporting was defined as EI/BMRest <1.2.

Results: Mean EI/BMR was 1.30 among butchers (BMI 24.6 ± 2.5), 1.33 among fishmongers (BMI 25.3 ± 2.0) and 1.32 among schoolteachers (BMI 25.3 ± 3.1) (P=0.716). Overall, the prevalence of under-reporters, EI:BMR <1.2, was 30%; the prevalence the 3 groups was butchers 44.4% (n=8), fishmongers 22.2% (n=4) and schoolteachers 22.2% (n=4). The difference in the prevalence of mis-reporters was only significantly different between groups when using a cut-off point <1.35 (P=0.018) but not <1.2. Mis-reporters were all non-smokers and heavier than acceptable reporters (BMI 28 vs 24; P= 0.03). The contribution of alcohol to total energy intake was lower among under-reporters (6 vs 12% as energy; P= 0.001) while there were no significant differences in fat, protein or carbohydrates.

Conclusions: There was no difference in prevalence of under-reporters using the usual cut-off point of 1.2. However a greater proportion of butchers were classified as under-reporters compared to fishmongers and schoolteachers using a EI:BMR <1.35 (P=0.018). Occupational food skills may not play an important role in reporting food intake. Alcohol seems to be under-reported in food under-reporting.

3.1 INTRODUCTION

Diet and nutrition are key factors in promoting and maintaining good health throughout life (WHO 2003). Information on food consumption is paramount to monitor dietary recommendations and thus contributing to implementation and to future policy making. However, mis-reporting of food consumption interferes with translation of nutritional understanding into practical application.

Dietary mis-reporting seems more prevalent in certain groups such as women and the obese. Recent evidence suggests that intentional dietary mis-reporting is substantial (McDiarmid and Blundell 1997; Lara *et al.*, 2004) and is associated with factors such as current and past dieting (Lara *et al.*, 2004), social desirability (Hebert *et al.*, 1997; Taren *et al.*, 1999), dietary restraint (McDiarmid and Blundell, 1997), disinhibition (Lara *et al.*, 2004), educational level, income, body mass, physical activity pattern, age, and smoking (Briefel *et al.*, 1997; Price *et al.*, 1997; Johansson *et al.*, 1998; Satia-abouta *et al.*, 2002).

The possibility that some population subgroups might be able to provide more accurate dietary data has scarcely been studied. Dietitians have been considered a group likely to provide accurate reports given their familiarity with dietary methodology. However, findings from these studies are not reassuring (Goris and Westerterp, 1999; Champagne *et al.*, 2002). Among non-professional people, food retailers may have certain advantages over people employed in other occupations. The closer contact with food through their occupation may endow them with better skills to estimate food portion size, knowledge about specific foods, and possibly more health-oriented lifestyles. In addition these groups may be influential in other people choices. The hypothesis that some groups employed in food retailing, i.e. butchers and fishmongers, may be able to

report food consumption more accurately than groups whose occupation is not related with food, was tested. These were compared to a group of schoolteachers, who are also considered influential but whose occupation and training does not involve contact with food.

3.2 METHODS

3.2.1 Subjects

Fifty-four subjects, 18 butchers, 18 fishmongers and 18 schoolteachers in Glasgow (Scotland) participated in this study. The butchers and fishmongers were identified through the "Yellow Pages". Retailers were contacted by telephone and a subsequent visit was arranged. The schoolteachers participating in this study were primary school teachers and were approached in person.

The inclusion criteria comprised subjects employed on a full time basis and with at least 5 years of experience in the present occupation and volunteering to participate in this study. This criterion was established in order to secure subjects with a long-term involvement and exposure to the food characterising their occupation in the case of the food retailers. Subjects with known clinical diagnosis following a special therapeutic diet, or subjects seeking weight management were excluded. Home economics teachers with a known knowledge in nutrition were excluded from this study. Although not an established criteria, participants in this study were all males. Participants accepting participation in this study were interviewed at their place of work.

3.2.2 Experimental design

On a single occasion participants completed a 2-page Dietary Targets Monitor (DTM) (Lean *et al.*, 2002) and a 7-d food diary [**Appendices 4-5**]. Participants recorded their food intakes during seven consecutive days including each day of the week, using a food diary. This dietary method was chosen on the assumption that seven days is considered the shortest period of time to get a picture of habitual food consumption of an individual, allowing for comparisons between weekend- and week-day patterns. A food diary estimating portion sizes instead of weighed records was chosen because it represents a lesser burden for the participant while still providing a good estimation (Bingham, 1987). In addition, it was assumed that subjects involved in these occupations might have better skill in estimating portion sizes given that weighing foods is a daily routine in their jobs. Special emphasis was placed on the importance of maintaining the usual eating patterns during the recording period e.g. number of meals or snacks per day or the type of foods usually eaten, consumption of salt at the table. The need to record food intake as soon as possible after each meal was stressed to avoid lapses of memory. Food records were checked for completeness and analysed using Diet 5 (Wisc, 2000).

3.2.3 Anthropometric measurements

Body weight, recorded in kilograms, was measured using a Salter portable digital scale (Salter Ltd, Kent); subjects were weighed without shoes but wearing light clothes; e.g. t-shirt and trousers because these measurements were carried out in their place of work. Waist circumference, in centimetres, was measured with an inelastic measuring tape at the midpoint between the lower rib and the iliac crest; and height, recorded in metres, was also measured without shoes using the Leicester portable stadiometer (Invicta

Plastics Ltd, Leicester). Anthropometric measurements were obtained using standard methods (WHO 1995).

3.2.4 Dietary mis-reporting

Dietary under-reporters were identified by means of the energy intake to basal metabolic rate ratio (EI:BMR) (Goldberg *et al.*, 1991). Basal metabolic rate was estimated with the equations recommended by the UK Department of Health (1991). Given the lack of information on physical activity levels, two different cut-off points were chosen to estimate the prevalence of mis-reporters. These were EI:BMR <1.2 and <1.35 since these have been the most commonly reported in the literature.

3.2.5 Statistical analysis

Descriptive statistics and one-way analysis of variance (ANOVA) were used for comparison between occupation groups (butchers, fishmongers and schoolteachers) taking a significance level of 0.05. A student t-test was used for comparing groups according to the EI:BMR ratios (e.g. <1.2 vs >1.2). Chi-square test was used to assess comparisons between categorical variables. All statistical comparisons were carried out with SPSS 10.5 for windows (SPSS Inc., Chicago, Ill, USA).

3.3 Results

The characteristics of the groups in the present study are shown in **table 3.1**. On average butchers were taller than fishmongers and schoolteachers; however mean BMI was the same. There were no significant differences in age. Overall the EI:BMR ratio was low but no significant difference was observed between groups.

The prevalence of a EI:BMR cut-off point <1.2 was 30% of all subjects. No significant difference for the comparison between groups was observed for this cut-off point. Fifty-two percent of all subjects presented an EI:BMR below 1.35. With this criterion, a lesser number of fishmonger and schoolteachers under-reported food energy intake compared with butchers (P=0.018) (**Table 3.2**).

Under-reporting was strongly associated with smoking status (P=0.002); never smokers (72%) were more likely to under-report than current (5.6%) and former smokers (11.1%) (**Table 3.3**).

The patterns of macronutrient intake between under-reporters and acceptable reporters are shown in **table 3.4**. Significant differences were observed for the consumption of alcohol between under-reporters and acceptable reporters defined by any of the two EI:BMR cut-off points.

Subjects reporting a EI:BMR <1.2 were heavier than those with a EI:BMR >1.2 (median BMI 27.2 vs 23.5; P=0.07).

Alcohol consumption estimated from the food records, whether expressed in kcals or as a percentage of total energy intake estimated from food records, was significantly related to the EI:BMR ratio during the recording week, after adjusting for BMI (**Figure 3.1**); such a relationship was not observed with alcohol consumption estimated from DTM. In addition, alcohol intake reported during the recording week was significantly lower than estimated at the beginning of the study by a DTM (-67 kcals; P=0.013) (**Figure 3.2**). This difference was also positively related to EI:BMR (**Figure 3.3**).

EI:BMR was not significantly related to dietary fat, carbohydrates or protein expressed as percentage of energy intake.

3.4 DISCUSSION

Dietary mis-reporting is a pervasive problem for most dietary surveys, particularly amongst the overweight and obese who now form more than half of the entire population.

Under the hypothesis that some subgroups might be more skilful and accurate in estimating food consumption dietitians were studied by Champagne at el. (2002), who showed a better reporting accuracy in their study of dietitians compared with non-dietitians. Goris and Westerterp (1999) in another study in dietitians found a mean under-reporting of energy intake of 16%. In this later study, under-eating accounted for all under-reporting i.e. no under-recording of foods seemed to be involved in these subjects. In a second study, Goris and Westerterp (2000) reported no evidence of under-reporting after confronting the dietitians with their results. An apparent accurate food recording led to the conclusion that under-reporting was not necessarily the result of a dishonest action; however factors such as social desirability or seeking of social approval might have been involved as shown in other studies (Hebert *et al.*, 1997). Other professions do not seem to have been studied.

In the present study the prevalence of dietary mis-reporters in a group of men employed in food-related occupations, fishmongers and butchers, varied with mis-reporters being more prevalent among butchers (44% with EI:BMR<1.2; and 78% EI:BMR <1.35) and similar among fishmongers and schoolteachers whose prevalence ranged from 33 to 44% (P=0.018)

Between groups differences in the prevalence of mis-reporters was observed only when using a more "tolerable" cut-off point; e.g. mis-reporting was less prevalent among

fishmongers when using a EI:BMR ratio <1.35, however the number were small and true differences cannot be excluded with confidence at other cut-offs.

In the present study it was not possible to discriminate between under-recording and under-eating given that body weight was only measured once.

Another finding in the present study was an indication for selective mis-reporting of alcohol intake in these men. As assessed by means of a FFQ at the beginning of the study these subjects reported similar usual intakes of alcohol, however a significantly lower consumption was reported during the recording week (P=0.01). Whether alcohol consumption was under-recorded or whether it was a real but under-representative consumption of the usual intake cannot be resolved. However the fact that other studies in men have suggested under-reporting of alcohol consumption seem to support the present findings (Briefel *et al.*, 1997). Goris *et al.* (2000) also reported a possible selective mis-reporting of dietary fat by obese men. The lack of biomarkers to validate other fat and carbohydrate intakes makes it impossible to corroborate these findings.

In the present study, data on alcohol consumption obtained through a DTM (a retrospective methods assumed to represent habitual consumption) was compared with that obtained from a 7-d food diary (current consumption) to assess possible bias in alcohol intake (habitual vs actual alcohol consumption). Results showed significant association between the bias in alcohol consumption and EI:BMR; thus suggesting the possibility that energy from alcohol may have been significantly under-reported.

In conclusion these results show that the possible skills acquired by subjects involved in food related occupation such as butchers and fishmongers do not influence the food reporting process and under-reporting of food consumption is common. A possible

selective under-reporting of alcohol consumption may be commonly present in male subjects.

3.5 New research questions

The data in this chapter suggest more accurate dietary reporting by fishmongers than schoolteachers and much better than butchers. This raises new questions about the actual dietary records of these groups and their influence on health.

1. Do butchers, fishmongers and schoolteachers report quantitatively different diet compositions?

2. Is there any relationship between diet composition and health indicators in these groups?

Table 3.1 Characteristics of the groups

	Butchers (n=18)	Fishmongers (n=18)	Schoolteachers (n=18)	P
Age (yrs)	48 (43, 53)	43 (39, 48)	47 (38, 52)	0.165
Weight (kg)	76.2 (71.1, 81.4)	75.7 (68.8, 82.5)	75.0 (68.2, 82.0)	0.526
Height (m)	1.78 (1.76, 1.80)	1.73 (1.70, 1.75)	1.75 (1.70, 1.79)	0.004
BMI (kg/m^2)	24.6 (23.2, 26.0)	25.3 (23.2, 27.4)	25.3 (23.5, 27.0)	0.544
EI:BMR	1.30 (1.20, 1.40)	1.33 (1.19, 1.47)	1.32 (1.18, 1.45)	0.716

Values are means (95% CI for mean)
P-value represents ANOVA analysis

Table 3.2 Under-reporters by two different cut-off points EI:BMR

	Butchers (n=18)	**Fishmongers (n=18)**	**Schoolteachers (n=18)**	**P**
	n (%)	**n (%)**	**n (%)**	
EI:BMR < 1.35	14 (78)	6 (33)	8 (44.4)	
EI:BMR > 1.35	4 (22)	12 (67)	10 (55.6)	*0.018
EI:BMR < 1.2	8 (44)	4 (22)	4 (22)	
EI:BMR > 1.2	10 (56)	14 (78)	14 (78)	**0.129

*Chi-square; χ^2=7.2
** Chi-square; χ^2=2.0

Table 3.3 Under reporting by smoking status

	Current smokers	**Former smokers**	**Never smokers**	***P**
	n (%)	**n (%)**	**n (%)**	
EI:BMR < 1.35	4 (22.2)	4 (22.2)	15 (83.3)	
EI:BMR > 1.35	14 (77.8)	14 (77.8)	3 (16.7)	*0.008
EI:BMR < 1.2	1 (5.6)	2 (11.1)	13 (72.2)	
EI:BMR > 1.2	17 (94.4)	16 (88.9)	5 (27.8)	**0.002

*Chi-square test; χ^2=9.63
**Chi-square test; χ^2=12.66

Table 3.4 Self-reported macronutrient intake by EI:BMR

	EI:BMR < 1.2	EI:BMR > 1.2	P	EI:BMR < 1.35	EI:BMR > 1.35	P
EI:BMR	1.09 (1.01, 1.18)	1.42 (1.33, 1.51)	0.000001	1.16 (1.09, 1.22)	1.50 (1.39, 1.61)	0.000001
Energy (kjoules)	6540 (6060, 7080)	8520 (7980, 9060)	0.000001	6960 (6540, 7320)	9000 (8340, 9660)	0.000001
% Energy						
Protein	17.5 (15.1, 19.4)	17.3 (14.9, 18.7)	0.867	17.7 (15.1, 19.3)	17.5 (14.7, 19.2)	0.843
Carbohydrate	42.1 (36.6, 45.6)	42.2 (37.2, 44.2)	0.680	43.5 (39.2, 45.9)	44.0 (38.6, 46.5)	0.653
Fat	40.4 (35.4, 44.2)	40.5 (35.6, 44.6)	0.879	38.8 (34.5, 41.7)	38.5 (33.5, 41.3)	0.889
Alcohol	5.6 (2.9, 8.3)	11.7 (9.5, 14.0)	0.001	7.2 (4.8, 9.6)	12.8 (10.2, 15.4)	0.002

Values are means (95% CI for mean)

P-values for student t-tests

Figure 3.1

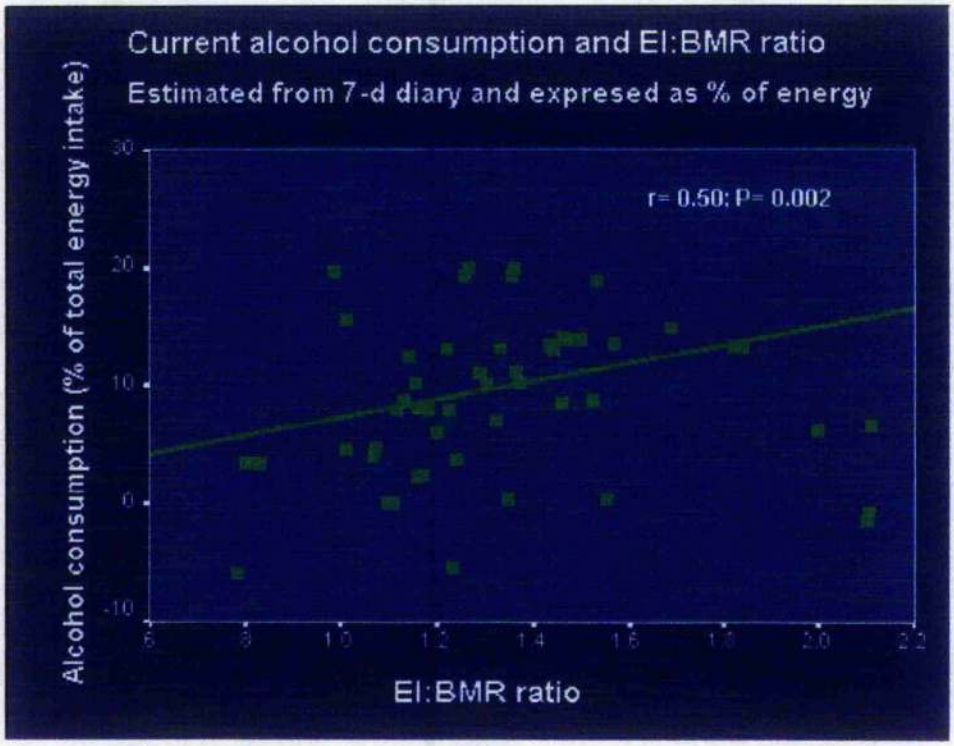

Current alcohol consumption and EI:BMR ratio
Estimated from 7-d diary and expresed as % of energy
r= 0.50; P= 0.002
Alcohol consumption (% of total energy intake)
30
20
10
0
-10
6
8
10
12
14
16
18
20
22
EI:BMR ratio

Figure 3.2

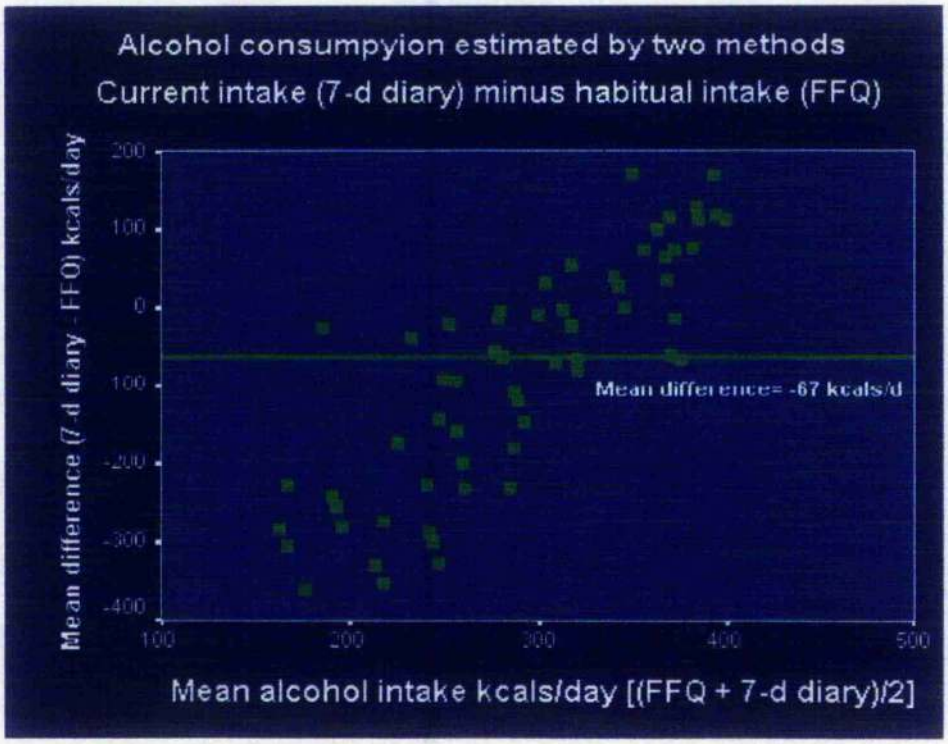
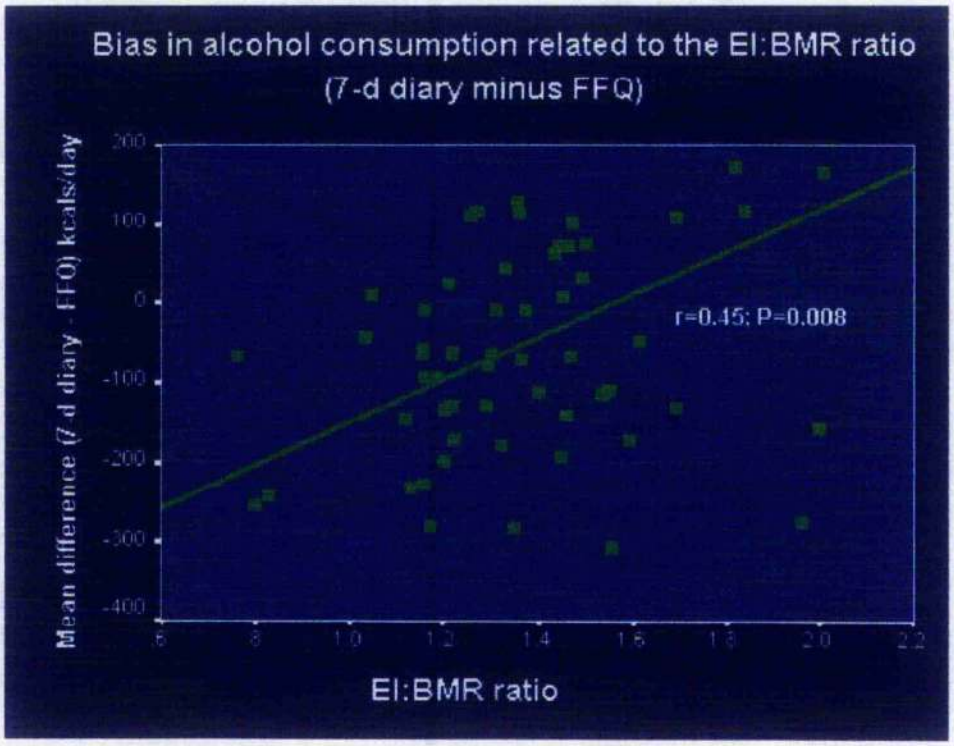

Alcohol consumpyion estimated by two methods
Current intake (7-d diary) minus habitual intake (FFQ)
Mean difference (7-d diary - FFQ) kcals/day
200
100
0
-100
-200
-300
-400
Mean difference= -67 kcals/d
100
200
300
400
500
Mean alcohol intake kcals/day [(FFQ + 7-d diary)/2]

Figure 3.3


Bias in alcohol consumption related to the EI:BMR ratio
(7-d diary minus FFQ)
Mean difference (7-d diary - FFQ) kcals/day
200
100
0
-100
-200
-300
-400
r=0.45; P=0.008
6
8
10
12
14
16
18
20
22
EI:BMR ratio

Chapter 4

Dietary practices, food choice and health in male subjects involved in food-related occupations

This chapter represents unpublished data, but it is written in the format intended for submission to Journal of Human Nutrition and Dietetics

4.0 Context of this chapter

This chapter describes a dietary analysis conducted to address the two research questions defined in section 3.5

Research questions

1. Do butchers, fishmongers and schoolteachers report quantitatively different diet compositions?

2. Is there any relationship between diet composition and health indicators in these groups?

ABSTRACT

Aim: To assess the dietary practices and its relation with blood pressure in men involved in food-related occupations.

Methods: Fifty four subjects, 18 butchers, 18 fishmongers and 18 schoolteachers (age 39-53 years, BMI 23.2-27.4 kg/m^2) completed a FFQ and a 7-d food diary. Under-reporting was defined as EI/BMRest <1.2.

Results: Assessment of eating habits of these groups showed that more butchers consumed semi-skimmed milk than fishmongers and schoolteachers. Overall, the target for white fish was achieved by 15% and for oil-rich fish by 9% of all subjects combined; fishmonger reached 90 and 100% success with these targets. Fishmongers also reported a greater consumption of sweets and chocolate, cakes, ice cream and biscuits but consumed eggs less frequently than the other groups. On average 30% of all subjects, combined, achieved the dietary target of five portions of fruit and vegetables per day; a significantly greater proportion of schoolteachers achieved the target than the other groups.

Systolic blood pressure among fishmongers was significantly lower (P=0.05) than among butchers and teachers independently of BMI and age. In addition, smoking and alcohol consumption was greater among butchers followed by fishmongers and schoolteachers (**Table 4.9**).

Conclusions: Some occupations positively influence the diet of individuals. In the present study, fishmongers were more likely to achieve the dietary target for fish in the Scottish population. The consumption of fish may have imparted these subjects with a protective phenotype against cardiovascular risk, i.e. lower blood pressure.

4.1 INTRODUCTION

4.1.1 Diet, health and occupation

A positive relationship between socioeconomic status and a healthier diet has been shown by most dietary surveys e.g. a higher consumption of fruits and vegetables. Occupation influences diet in different ways. Level of income determines availability of economic and material resources influencing dietary intake by limiting access to food. Occupation also influences dietary intake by influencing energy expenditure. It may provide also an environment likely to influence social identity through work-based culture and social networks (Galobardes *et al.*, 2001). In addition occupation features such as working hours, time allowed to consume meals and catering facilities are likely to affect food choice.

4.1.2 Relationship of occupation to food choice

The food industry is a major employer and people employed in several food-related occupations are likely to have particular influence over the consumer's food choices (Scottish Office, 1996). Food retailers have daily contact with customers and may be likely to influence public health dietary interventions, as well as the diet of their own families. It is possible that they are more aware of food-health links, but given the possible lack of training in nutrition in these groups, their beliefs about food and health may be different from the current dietary recommendations and potentially biased toward their own occupational expertise or heavily influenced by popular beliefs (e.g. meat is food for men). Whether or not these groups differ in their beliefs and attitudes toward food and nutrition, or in dietary practices has not been previously explored.

These aspects are important in terms of understanding the food choice patterns, dietary behaviour and therefore nutrient intake, as well as in establishing health preventive policies in these individuals.

This study compares the diets of groups employed in food retailing, i.e. butchers and fishmongers, and their relationships with blood pressure, BMI. A group of schoolteachers who are also considered an influential group was studied for comparisons but do not have occupational involvement with food.

4.2 Methods

4.2.1 Subjects

Eighteen butchers, eighteen fishmongers and eighteen schoolteachers in Glasgow, Scotland completed this study. This sample has already been described in chapter 3 (3.2.1).

4.2.2 Experimental design

On a single occasion participants completed a short DTM (Lean *et al.*, 2002) **[Appendix 4]**.

A questionnaire used in the Pan-European survey of consumer attitudes to food, nutrition and health (Gibney *et al.*, 1997) was used in this study **[Appendix 6]**. Subjects were asked to complete the questionnaire during day one of the study, usually at their place of work.

4.2.3 Anthropometric measurements

This information has been described in chapter 3 (3.2.3)

4.2.4 Blood pressure

An Omron automatic digital blood pressure monitor was used to measure blood pressure. During a morning visit, duplicate blood pressure measurements were obtained on the right arm following a simple routine procedure (i.e. asking the participant to remove outer garments and sitting quietly for 5 minutes prior to the measurement; positioning the colour mark of the cuff over the brachial artery; participant remaining quiet and relaxed during measurement). The average value for blood pressure is reported in the results.

4.2.5 Statistical analysis

Descriptive statistics and ANOVA analysis were used for comparison between groups taking a significance level of 0.05. Adjustments were performed for BMI and age differences between these groups. Chi-square test was used to compare categorical variables. All statistical comparisons were carried out with SPSS 9.0 for windows (SPSS Inc., Chicago, Ill, USA).

4.3 Results

Among the physical characteristics of the groups studied, only a significant difference in height was observed with the butchers being the taller group (**Table 4.1**). Butchers

reported the longest duration in their occupation. Most of the participants were married (76%); 14% were single and 10% divorced (**Table 4.2**).

Assessment of eating habits of these groups showed significant differences in the type of milk consumed; a higher proportion of butchers were more inclined to use low-fat milk (**Table 4.3**).

Comparisons of the weekly consumption of particular food groups revealed significant differences in the consumption of white and oil-rich fish, more frequently consumed by fishmongers with similar consumptions in butchers and schoolteachers. Fishmongers also reported a greater consumption of sweets and chocolate, cakes, ice cream and biscuits but consumed eggs less frequently than the other groups (**Table 4.4**).

On average, 30% of all subjects achieved the dietary target of five portions of fruit and vegetables per day; a significantly greater proportion of schoolteachers achieved the target than the other groups. The average figure for all subjects achieving the target for white fish was 15% and for oil-rich fish was 9%. Fishmonger reached 90 and 100% success with these targets (**Table 4.5**).

The influences on food choice among these groups were particularly related to the "quality or freshness of foods"; three quarters of butchers and half of schoolteachers. "Decisions or preferences of partner" together with "taste of food" and "convenience in preparation" prevailed among fishmongers (**Table 4.6**).

Butchers reported mass media as the main source of information on nutrition, while information on food packages was reported by schoolteachers. Fishmongers reported mass media and information provided by supermarkets in similar rates (**Table 4.7**).

Among the perceived difficulties to eat healthy, irregular working hours prevailed among fishmongers while a "busy lifestyle" was the main problem among schoolteachers (**Table 4.8**).

Assessment of blood pressure showed that fishmongers had a significantly lower systolic blood pressure (P=0.05) and lower but significantly different pulse rates than butchers and teachers. In addition, smoking and alcohol consumption was greater among butchers followed by fishmongers and schoolteachers (**Table 4.9**).

4.4 Discussion

The present study compared the dietary habits of two groups of food retailers, fishmongers and butchers, with those of schoolteachers. All these groups are considered potentially influential on other people's food choice e.g. customers and pupils. No similar studies are available on these particular occupational groups and proper comparisons are thus not possible.

The present study confirmed the hypothesis that some population subgroups may show different dietary patterns influenced by aspects of social status in extraordinary ways. Occupation seems to be a factor that may have influenced positively the consumption of fish among fishmongers. It is possible that simply day-to-day exposure to a particular food may influence subject's perception. In addition, it is possible that social access to fish, possibly at lower prices, may be another explanation. In addition, occupation may provide also an environment likely to influence social identity through work-based culture and social networks (Galobardes *et al.*, 2001). However, it is important to consider that other factors may have influenced these results since no significant differences were observed in the consumption of meat or meat products by butchers.

Consumption of fish is associated with reduced risk for coronary heart disease (CHD) and mortality (Kromhout *et al.*, 1985). There is evidence indicating that one of the beneficial effects of fish consumption is given by a lower blood pressure and heart rate values. Paulctto *et al.* (1996) reported lower systolic and diastolic blood pressure (10 and 4 mmHg respectively) among Tanzanian fish-eaters compared with those consuming a vegetarian diet. Consumption of fish by these subjects was extraordinarily high (300 to 600 g/day). There is a lack of experimental studies on the effects of fish consumption in healthy subjects. von Houwelingen *et al.* (1987) reported no significant difference in blood pressure changes in healthy volunteers after eating 100 g of either mackerel or meat during six weeks. However, these authors reported a decrease in the compliance with time, particularly when eating mackerel.

In the present study, a significantly lower systolic blood pressure was observed among fishmongers. This difference was independent of BMI, age, smoking, and recent weight change. The groups included in the present study differed in the consumption of both types of fish, white and oil-rich. All fishmongers reported eating fish, with up to two thirds eating oil-rich fish, and 90% eating white fish, at least once a week. Thus a greater proportion of fishmongers achieved the dietary target for the Scottish population of fish of 1-2 portions per week (SHS, 1998). Contrastingly, up to 40% of butchers and teachers reported not eating oil-rich fish on a weekly basis.

The influences in food choice among these subjects were not different from other population groups (Lennernas *et al.*, 1997; Gibney *et al.*, 1997). Busy lifestyles, irregular working hours, were commonly reported as barriers to eat healthy among these groups.

Veracity of self-reported data is always an issue of concern. However in observational/ epidemiological studies it is sometimes the only way to obtain information in big scale. In addition, the groups included in this study are not representative of their group of origin. However given the diminishing number of people in food-related occupation, it is rather difficult to obtain a representative one. Efforts were made to secure the inclusion of people working full time in these occupations and with at least 5 years of experience in their job.

An improvement on endothelial function by omega-3 fatty acids in oil-rich fish is one proposed mechanism involved in the lowering of blood pressure (Connor 2000). The mechanism involved is attenuation in the responses of vascular resistance and blood flow to angiotensin, i.e. less vascular reactivity. Specifically EPA is the precursor of a series of eicosanoids, thromboxanes and Leukotrienes, which reduce platelet aggregation and increase vasodilation. This could account in part for those fish oil effects that may lead to reduced clotting activity and decreased blood pressure

Therefore given that consumption of only small amounts of fish are necessary to provide protection against cardiovascular risk, it is quite possible that, indeed, fishmonger's lower blood pressure was given by a regular long-term consumption of fish. Epidemiological evidence suggests that consumption of meat, but not fish (or fruits and vegetables), was significantly associated with higher increases in blood pressure over a 7-year period. (Miura *et al.*, 2004)

In conclusion, it is likely that some occupations may positively influence the diet of individuals. In the present study, fishmongers reported consuming fish on a regular weekly basis with most subjects achieving the dietary target for fish in the Scottish

population. The consumption of fish may have imparted these subjects with a protective phenotype against cardiovascular risk, i.e. lower blood pressure and possibly other metabolic factors such as blood lipids, inflammatory factors, or insulin sensitivity. These results justify the design of experimental studies assessing the benefits of fish consumption in healthy subjects and hypertensive patients.

4.5 New Research questions

Although increasing fish consumption is a dietary target in Scotland and USA there is unbelievably little direct evidence.

The results from the study described in this chapter with greater consumption of fish and lower blood pressure in fishmongers leads to new research questions to be addressed in the next chapter.

1. Does higher consumption of fish reduces blood pressure?

2. Does higher consumption of fish modify other indices of metabolic syndrome?

Table 4.1 Physical characteristics

	Butchers (n=18)	Fishmongers (n=18)	Schoolteachers (n=18)	p
Duration in current occupation (yrs)	25 (10, 31)	17 (7, 24)	15 (6, 20)	0.381
Age (yrs)	48 (43, 53)	43 (39, 48)	47 (38, 52)	0.165
Weight (kg)	76.2 (71.1, 81.4)	75.7 (68.8, 82.5)	75.0 (68.2, 82.0)	0.526
Height (m)	1.78 (1.76, 1.80)	1.73 (1.70, 1.75)	1.75 (1.70, 1.79)	0.004
BMI (kg/m^2)	24.6 (23.2, 26.0)	25.3 (23.2, 27.4)	25.3 (23.5, 27.0)	0.544

Values are mean ± SD. P-values correspond to ANOVA Analysis

Table 4.2 Marital status

	Butchers (n=18)	Fishmongers (n=18)	Schoolteachers (n=18)
	n (%)	n (%)	n (%)
Single	2 (11)	3 (17)	3 (17)
Married	14 (78)	13 (72)	15 (83)
Divorced	2 (11)	2 (11)	0 (0)

Table 4.3 Eating habits monitor (EHM)

Type of food	Butchers (n=18) n (%)	Fishmongers (n=18) n (%)	Schoolteachers (n=18) n (%)	Chi square P-value
Bread: White	14 (67)	12 (80)	13 (87)	
Wholemeal	4 (22)	3 (20)	2 (13)	0.800
Spread: Butter	12 (67)	12 (80)	12 (80)	
Soft margarine	4 (22)	3 (20)	3 (20)	
Reduced/low fat spread	2 (11)	0 (0)	2 (13)	0.732
Milk: Whole	8 (44)	12 (80)	13 (87)	
Semi-skimmed	10 (56)	3 (20)	2 (13)	0.018

Table 4.4 Portions consumed per week (EHM)

Food	Fishmongers (n=18)	Butchers (n=18)	Schoolteachers (n=18)	p
Breakfast cereals	4.1 ± 6.4	2.1 ± 2.9	3.1 ± 2.5	0.326
Fruit	6.6 ± 5.9	8.9 ± 8.1	7.9 ± 8.3	0.422
Cooked green vegetables	4.8 ± 1.9	5.9 ± 4.9	4.9 ± 4.8	0.348
Cooked root vegetables	4.3 ± 2.5	4.9 ± 5.0	4.0 ± 5.2	0.621
Raw vegetables	1.1 ± 0.9	2.1 ± 2.0	1.5 ± 2.3	0.129
Chips	3.1 ± 1.4	3.9 ± 5.2	3.5 ± 5.1	0.620
Potatoes/pasta/rice	6.4 ± 5.7	4.6 ± 2.3	5.0 ± 2.5	0.227
Meat	4.4 ± 2.3	3.7 ± 1.4	4.1 ± 1.4	0.322
Meat products	4.1 ± 1.7	4.0 ± 2.3	4.0 ± 2.2	0.908
Poultry	2.3 ± 1.0	2.1 ± 1.1	2.2 ± 1.2	0.476
White fish	2.0 ± 1.8	0.5 ± 0.3	0.7 ± 0.2	0.002
Oil-rich fish	1.1 ± 0.9	0.2 ±0.2	0.3 ±0.3	0.001
Cheese	3.3 ± 2.9	3.3 ±1.9	3.5 ±1.8	0.975
Beans/pulses	1.5 ± 1.2	1.3 ± 1.0	1.0 ± 1.0	0.712
Sweets, chocolates	9.1 ± 6.6	2.1 ± 2.4	2.5 ± 2.5	0.001
Ice cream	1.1 ± 0.9	0.4 ± 0.4	0.8 ± 0.3	0.025
Crisps, savoury snacks	3.0 ± 1.6	4.2 ± 5.5	3.5 ± 5.1	0.459
Fruit juice	11.5 ± 15.4	6.9 ± 6.3	7.0 ± 6.4	0.267
Soft drinks	5.0 ± 6.1	2.4 ± 2.6	3.0 ± 2.5	0.185
Cakes, scones	5.6 ± 5.9	0.9 ± 1.8	2.1 ± 1.7	0.006
Biscuits	11.5 ± 10.8	2.5 ± 2.6	3.2 ± 2.8	0.003
Eggs	0.9 ± 1.1	2.4 ± 2.0	2.1 ± 2.2	0.012

Values are mean ± SD. P-values correspond to ANOVA Analysis

Table 4.5 Achieving dietary targets (EHM)

	Portions	Butchers (n=18) n (%)	Fishmongers (n=18) n (%)	Schoolteachers (n=18) n (%)	SHS 1998 %
Fruit + fruit juice + Green/ root & raw Vegetables	5/day or more	4 (22)	5 (28)	7 (39)	
	4/day	2 (11)	4 (22)	5 (28)	
	3/day	4 (22)	3 (17)	4 (22)	
	2/day	4 (22)	3 (17)	1 (5.6)	
	1/day	4 (22)	3 (17)	1 (5.6)	
White fish	≥ 2/wk	0 (0)	7 (39)	1 (5)	15
	1/wk	4 (18)	9 (50)	5 (28)	41
	0.1-0.9/wk	12 (67)	2 (11)	8 (62)	25
	0	2 (11)	0 (0)	1 (5)	18
Oil-rich fish	≥ 2/wk	0 (0)	5 (28)	0 (0)	16
	1/wk	0 (0)	7 (39)	1 (5)	23
	0.1-0.9/wk	10 (56)	6 (33)	10 (56)	27
	0	8 (44)	0 (0)	7 (39)	33

SHS= Scottish Health Survey

Table 4.6 Important influences on food choice

	Butchers (n=18)	Fishmongers (n=18)	Schoolteachers (n=18)
	n (%)	n (%)	n (%)
Quality of freshness of food	14 (78)	0 (0)	9 (50)
What family/spouse/partner will eat	0 (0)	6 (33)	0 (0)
Trying to eat a healthy/balanced diet	4 (22)	2 (11)	5 (28)
Taste of food	0 (0)	5 (28)	2 (11)
Convenience in preparation	0 (0)	5 (28)	2 (11)

Table 4.7 Sources of information on healthy eating (Subjects were asked to provide as many answers as appropriate)

	Butchers (n=18)	Fishmongers (n=18)	Schoolteachers (n=18)
	n (%)	n (%)	n (%)
Programmes/news, advertising on TV/radio	14 (78)	6 (33)	3 (17)
Newspapers, magazines	10 (55.6)	6 (33)	4 (22)
Information on food packages	4 (22)	0 (0)	10 (78)
Supermarkets	0 (0)	6 (33)	5 (28)

Table 4.8 Perceived difficulties to eat healthy

	Butchers (n=18)	Fishmongers (n=18)	Schooltcachers (n=18)
	n (%)	n (%)	n (%)
Irregular working hours	4 (22)	10 (55)	0 (0)
Busy lifestyle	6 (33)	6 (33)	15 (83)
Unappealing food	4 (22)	0 (0)	2 (11)
Giving up foods I like	4 (22)	4 (22)	3 (17)
Willpower	0	4 (22)	0

Table 4.9 Blood pressure

	Butchers (n=18)	Fishmongers (n=18)	Schoolteachers (n=18)	p
Systolic BP (mmHg)*	137 (132, 140)	130 (122, 129)	134 (129, 135)	0.050
Diastolic BP (mmHg)*	89 (84, 91)	84 (80, 86)	88 (82, 91)	0.158
Pulse (beats/min)*	78 (70, 83)	72 (65, 77)	74 (70, 79)	0.415
Alcohol (units/wk)*	37 (25, 48)	22 (16, 35)	11 (6, 15)	0.027
Smoking (cigarettes/day)	28 (19, 36) (n=8)	18 (15, 21) (n=7)	10 (6, 14) (n=5)	0.043

Values are mean (95% CI for the mean) P-values correspond to ANOVA Analysis

* Results for the comparisons of these variables were adjusted for BMI and age

Chapter 5

Benefits of salmon eating on traditional and novel vascular risk factors in young, non-obese healthy subjects

This work has been submitted for publication as Lara JJ, Economou M, Wallace AM, Rumley A, Lowe G, Slater C, Caslake M, Sattar N, Lean MEJ. Benefits of salmon eating on traditional and novel vascular risk factors in young, non-obese healthy subjects (submitted)

5.0 Context of this chapter

This chapter describes an intervention experimental study conducted to address the two research questions defined in section 4.5

Research questions

1. Does higher consumption of fish reduces blood pressure?

2. Does higher consumption of fish modify other indices of metabolic syndrome?

ABSTRACT

BACKGROUND Epidemiological evidence supports an association between consumption of oil-rich fish and a lower risk for cardiovascular diseases. The present experimental study tested this hypothesis in relation to CHD risk factors.

METHODS Forty-eight (16 men) non-obese, healthy adults aged 20 to 55yrs, consumed 125 grams/day of salmon for a four-week period followed by a four-week period with no-fish. Subjects were instructed to maintain their usual eating habits and physical activity patterns during the period of study to avoid body weight changes. Blood pressure, anthropometric, body composition, eating behaviour, and dietary information with fasting blood samples to determine traditional and novel risk markers and plasma fatty acids were obtained before and after each intervention period.

RESULTS Stable body weight and composition supported negligible changes in energy intake and physical activity in this study. Compared to no-fish, eating salmon significantly decreased systolic and diastolic as well as mean arterial blood pressure by 4%, fasting triglycerides by 15%, VLDL by 17% and LDL by 7%, and significantly increased HDL-cholesterol by 5% ($p<0.05$). Eating salmon significantly increased HDL-2 but not HDL-3, and a significantly decreased both, VLDL-1 and VLDL-2 cholesterol sub-fractions.

The changes in blood pressure and lipids alone with salmon intake predict around a 25% reduction in CHD risk based on the PROCAM risk calculator. Plasma adiponectin was 8.39 μmol/l with salmon and 7.52 with no-fish ($P=0.086$). No significant changes were found either in fasting plasma leptin, glucose or insulin after salmon consumption.

CONCLUSIONS Daily consumption of salmon improves traditional but not novel risk predictors for CHD in non-obese subjects.

5.1 INTRODUCTION

5.1.1 Fish consumption

Typically, fish is eaten once or twice a week and on average it provides about 20-30 kcal per capita per day or about 1% of dietary energy. However it may contribute up to 180 kcal per capita per day in a few countries where there is a lack of alternative protein foods produced locally or where there is a strong preference for fish (examples are Greenland, Iceland, Japan and some small island states) (WHO, 2003). Fish is favoured in human nutrition, partly as a low saturated fat alternative to meat. Fish is also a rich source of n-3 (omega-3) fatty acids, important structural components of the phospholipid membranes of tissues throughout the body and especially rich in the retina, brain, and spermatozoa (Connor, 2000). Omega-3 fatty acids consumption appears to be protective against coronary heart disease (CHD) (Hu & Willet, 2002). Therefore an increase in the consumption of fish has become a major target for dietary recommendations (Scottish Office, 1996; WHO, 2003). However, very few experimental studies have addressed fish consumption directly. Two small studies in healthy male volunteers have shown the benefits of consuming increasing fish consumption. Thorngren and Gustafson (1981) showed that eating oil-rich fish (mackerel and salmon) providing 2 to 3 g of eicosapentaenoic acid (EPA) per day during eleven weeks prolonged bleeding time by 42% and decreased platelet aggregability in 10 healthy men. Another study, in nine normolipidemic men, Lindgren *et al.*, (1991) showed that consuming salmon providing 2.1% of calories from n-3 fatty acids (20 and 22 carbon), during 40 days decreased

significantly plasma triglycerides while increasing HDL-cholesterol ($P < 0.01$); decreases in VLDL-cholesterol were not statistically significant.

5.1.2 Evidence of the benefits of fish consumption on cardiovascular disease

Twenty-five years ago a low risk for cardiovascular disease among Greenland Eskimos was linked with their high consumption of seafood (Dyerberg & Bang, 1979; Bang *et al.*, 1980). Since then, epidemiological and experimental evidence consistently suggests that consumption of fish is associated with reduced CHD risk and mortality (Kromhout *et al.*, 1985; Daviglus *et al.*, 1997). Omega-3 fatty acids provide a mechanism as they have been shown to have anti-atherogenic properties related to atherosclerosis, thrombosis and lipid metabolism (Connor 2000). In the only relevant experimental study, advice to eat oil-rich fish or fish oil supplements was found to reduce second myocardial infarction at 2 years (Burr *et al.*, 1989) but longer follow-up showed low effect (Ness *et al.*, 2002). However, it is possible that a lack of effect might be due to the fact that this study was following withdrawal of treatment. A report by the GISSI-Prevenzione group (1999), similar decreases in myocardial infarction and death were observed with n-3 PUFA supplements (1g/day) for 3.5 yrs. A recent meta-analysis by Bucher et al., (2002), concluded that consumption of dietary and non-dietary n-3 PUFAs may reduce mortality due to myocardial infarction, sudden death and overall mortality, in patients with CHD.

5.1.3 Biological effects of fish and/or fish oil

5.1.3.1 Blood lipids

Fish (with fish oil supplements) has been shown to influence blood lipids (Fehily *et al.*, 1983) and to reduce mortality after myocardial infarction (Burr *et al.*, 1989). Dietary n-3 fatty acids lower triglyceride and cholesterol concentrations (Connor, 2000). LDL cholesterol has been shown to increase with high doses of n-3 supplementation, particularly in individuals with elevated triglycerides but not so in normolipidaemic subjects; while increases in HDL-cholesterol concentrations are also observed (Connor 2000). The effects on triglycerides and HDL may suggest a mechanism involving insulin sensitivity.

5.1.3.2 Blood haemostasis

There is evidence showing that fish oil supplementation influences coagulation and fibrinolysis pathways (Connor, 2000). Fibrinogen is consistently associated with risk for CHD, while elevations in factor VII(c) and fibrinogen have been found to be a preceding factor to thrombosis and myocardial infarction in men (Meade *et al.*, 1980; 1986; Wilhelmsen *et al.*, 1984; Kannel *et al.*, 1987); as well as a major determinant of thrombosis severity (Heinrich *et al.*, 1994). FVII is associated with non-fasting plasma cholesterol (Meade *et al.*, 1980) and triglycerides (Miller *et al.*, 1985), suggesting an influencing role of dietary fat and probably other dietary components on haemostasis (Miller *et al.*, 1986).

5.1.3.3 Leptin

Recently, the consumption of fish has been associated with lower serum leptin, suggesting that fish might contribute to the regulation of this hormone (Winnicky *et al.*, 2002). Leptin

is an adipose tissue hormone involved in the development of obesity but also in the development of risk for CHD. Plasma leptin levels are higher in obese compared to normal weight people. However, African (Tanzanian) villagers whose diets included daily consumption of fish had lower levels of leptin than subjects whose diet was based on the consumption of vegetables without a difference in weight, suggesting greater leptin sensitivity in fish eaters (Winnicky *et al.*, 2002). Results were similar for men and women. Fish-eating women had lower leptin levels than non-fish-eating men, a surprising finding given that leptin levels in women are normally higher than in men. These results were shown in subjects eating between 300 and 600 g of fish per day.

A recent report, unavailable at the time we started the present study, (Mori et al., 2004) assessed whether consumption of fish enhanced the effects of weight loss on serum leptin levels in overweight treated hypertensive men and women. Subjects were randomized to a daily fishmeal, a weight-reduction regimen, the two regimens combined or a control group for 16 weeks. A significant greater change in serum leptin was observed in combination (fish + weight loss) group.

5.1.3.4 Blood pressure

In observational studies, high fish consumption has been associated with lower blood pressure and heart rate (Pauletto *et al.*, 1996: Dallongeville *et al.*, 2003). An improvement in endothelial function by omega-3 fatty acids in oil-rich fish is one proposed mechanism involved in the lowering of blood pressure. Experimental studies using fish oil supplements

support a dose-dependent effect of n-3 fatty acids on blood pressure but only in hypertensive and not in normotensive subjects (Morris *et al.*, 1993; Sacks *et al.*, 1994).

5.1.3.5 Adiponectin

Adiponectin, a cytokine-related hormone released in the adipose tissue, is considered a central factor in regulating many of the physiologic pathways controlling lipid and carbohydrate metabolism, and to mediate various vascular processes (Havel, 2004). Despite its origin in adipose tissue, adiponectin levels in plasma are paradoxically low in obese subjects, particularly in insulin-resistance states including metabolic syndrome and diabetes, as well as hypertension and coronary artery disease. Current evidence supports important anti-inflammatory and anti-atherogenic properties for adiponectin thus making it a potential target for the management and prevention of metabolic syndrome.

5.1.3.6 Inflammatory markers

ICAM-1 is a member of an immunoglobulin-like super family of proteins. The major known functions of ICAM-1 relate to its role in cell adhesion and migration. ICAM-1 is a counter-receptor for the ß2 leukocyte integrins LFA-1 ({alpha} Lß2, CD11a/CD18) and MAC-1 ({alpha} Mß2, CD11b/CD18), and their engagement results in leukocyte adhesion and transmigration through EC. There is strong evidence that the concentration of intercellular adhesion molecule-1 (ICAM-1) correlates positively with future cardiovascular risk in healthy men (Ridker *et al.*, 1998).

5.1.4 Aim of the present study

The present study tested the hypothesis that oil-rich fish may modulate both traditional (BP, lipids, glucose) and novel markers (adiponectin, leptin, inflammatory factors) of cardiovascular risk, in the absence of weight change or other changes in diet composition in a prospective trial in young non-obese, healthy men and women. Frozen salmon was chosen as a versatile and widely enjoyed fish. However, recognising that compliance and dietary reporting are so often problems, plasma DHA and EPA were measured as biomarkers of consumption. Four-week intervention periods providing 125 g of salmon daily were chosen after pilot work to establish volunteer compliance levels, although it is recognised that some effects on leptin and other markers may need longer exposure or a higher dosage than is possible within this practical constraint.

5.2 METHODS

5.2.1 Subjects

Forty-eight adult non-obese (Body Mass Index 18.5 to 29.9 kg/m^2) men and women were recruited by local advertising through the internet website of Glasgow University as well as using invitation letters to the staff at the Glasgow Royal Infirmary during the period between January-March, 2004. Participants in this study were healthy subjects. Therefore subjects with a previous diagnosis, taking any prescribed medication or following a special diet were excluded from this study.

Women were studied during the first phase of their menstrual cycle (i.e. blood samples were obtained early during their menstrual phase) or at any time if taking contraceptive pills. This criterion was established because this study aimed to study changes in leptin concentrations, which vary during the menstrual cycle.

The Glasgow Royal Infirmary Research Ethics Committee approved this study. After describing the procedures, signed consent was obtained from all participants.

5.2.2 Experimental design

A baseline assessment included dietary analysis, height, weight, waist, pulse, blood pressure and blood sampling. Participants provided retrospective and prospective dietary and eating behaviour information at the beginning and during each intervention period. Socio-economic, contraceptive medication and demographic data were also obtained.

Thereafter baseline assessments on their habitual diets were carried out and participants were then instructed to undertake an 8-week study period following a standard diet (Providing approximately 1.5 times participants' basal metabolic rate (UK, Department of Health, 1991), 50 % CHO, 35 % Fat, 15% protein). An experienced dietitian (J.L.) provided dietary advice with a portion exchange system for the meat/egg/fish components of meals. During the first 4-week period they consumed 125 g/day of oil-rich fish (salmon) followed by a control period (4-weeks) without fish. Diet composition remained 50% carbohydrates, 35% fat, 15% protein. The amount of daily salmon provided approximately 13.6 g of fat comprising 2.4 g of eicosapentaenoic acid (EPA) and docosahexaenoic acid

(DHA) (0.8 and 1.6 g, respectively). During the no-fish period, subjects were instructed particularly to avoid any kind of fish and to choose alternative options such as turkey, chicken, and cheese.

All subjects consumed the salmon before the no-fish period. The order was not randomised because the duration of any metabolic effects from salmon was not known and it was felt best to standardise the order which would result in a conservative estimate of the effect size (if there was a carry-over effect from salmon into the no-fish period) but with lower variance for differences than if the order was varied. Furthermore, given that participants did not always guarantee participation for more than one month, consumption of fish in the first phase would secure at least before and after comparisons in a large enough sample. In this event, all 48 subjects completed the salmon intervention period, but only 41 completed the no-fish period.

5.2.2.1 Seven-day food intake inventory

Participants recorded their food intakes during seven non-consecutive days including each day of the week, using a standard food diary [**Appendix 5**]. The reason for recording food intake in 7 non-consecutive rather than consecutive days was as an attempt to better characterise any variability of their diet during the 4-weeks periods. Food intake was recorded while subjects were following their habitual diets, during the fish-consumption and no-fish periods. This dietary method was chosen on the assumption that seven days is considered the shortest period of time to get a picture of habitual food consumption of an individual, allowing for comparisons between weekend- and week-day patterns. A food

diary estimating portion sizes instead of weighed records was chosen because it represents a lesser burden for the participant while still providing an acceptable estimate (Bingham, 1987). In addition, this method provides an estimation of total energy intake; this permits assessment of dietary mis-reporting. Participants were carefully instructed to record food portion sizes using household measures or provide label information when consuming packaged foods. Written instructions were provided. Special emphasis was placed on the importance of maintaining eating patterns during the recording period e.g. number of meals or snacks per day or the type of foods usually eaten, consumption of salt at the table. The need to record food intake as soon as possible after each meal was stressed to avoid lapses of memory. Food records were checked for completeness and analysed using Diet 5 (Wise, 2000).

5.2.2.2 Habitual eating patterns

A short food frequency questionnaire used in the 1998 Scottish Health Survey, the Scottish Dietary Targets Monitor (Lean *et al.*, 2003), designed specifically to assess dietary targets including fish consumption was administered at baseline to assess habitual diet [**Appendix 4**]. This was considered complementary to the food consumption data gathered by the 7-day food diaries.

5.2.2.3 Physical activity

Physical activity modifies beneficially most cardiovascular risk factors (Zimmet *et al.*, 1991). There is no ideal "gold-standard" method to measure physical activity, which

captures all types of activity. Self reported physical activity may be open to bias but it consistently reflects cardiovascular health (Zimmet *et al.*, 1991). A short questionnaire used in the 1998 Scottish Health Survey (Scottish Office, 1998) was employed to assess the physical activity patterns in this study **[Appendix 7]**. The purpose of assessing physical activity in this study was to characterise subject's patterns, principally to encourage maintenance of similar patterns during the study. Special emphasis was placed on the importance of maintaining their usual physical activity and eating habits during the study in order to maintain their body weight.

5.2.2.4 Cognitive measures

The three-factor eating (3-FEQ) questionnaire to assess dietary restraint, disinhibition and hunger (Stunkard and Messick, 1985) was used in this study [Appendix 3]. The 3-FEQ, also known as the eating inventory, is a 51-item instrument to measure eating behaviour developed in lean and obese people. It includes three scales or factors with high reliabilities (derived from factor analysis); factor I indicating cognitive or dietary restraint (21 items), factor II representing disinhibition of control (16 items), and factor III indicating perceived hunger (14 items). Thirty-six items require answers provided in a true-false basis whereas the other 15 employ a Likert scale for frequency. The range of possible scores ranges from 0 to 21 for restraint, from 0 to 16 for disinhibition, and from 0 to 14 for hunger, with higher scores indicating higher measures as described by Stunkard & Messick (1985).

5.2.2.5 Body composition and anthropometric measurements

Body composition was determined by air displacement plethysmography with the BODPOD system (Dempster and Aitkens, 1995). Body weight recorded in kilograms, was measured using a Salter portable digital scale without shoes and wearing a swimming costume (Salter Ltd, Kent); waist circumference, in centimetres, was measured with an inelastic measuring tape at the mid-point between iliac crest and lower rib; and height, recorded in metres, by using the Leicester portable stadiometer (Invicta Plastics Ltd, Leicester). All these measurements were obtained after an overnight fasting with the volunteers wearing a light swimming costume required for the BODPOD measurement. Anthropometric measurements were obtained using standard methods (WHO 1995).

5.2.2.6 Blood pressure

An Omron automatic digital blood pressure monitor was used to measure blood pressure. During a morning visit, duplicate blood pressure measurements were obtained on the right arm following a simple routine procedure (i.e. Asking the participant to remove outer garments and sit quietly for 5 minutes prior to the measurement; positioning the colour mark of the cuff over the brachial artery; participant remaining quiet and relaxed during measurement).

To minimise inter-observer variability, a single observer (J. Lara) obtained all data: dietary, body composition, anthropometric measurements, blood sampling and plasma separation, and measurement of blood pressure.

5.2.2.7 Blood sampling and biochemistry

An overnight fasting blood sample (~30 ml) was obtained at the beginning and at the end of each four-week period. Venepuncture was performed with a green 21 gauge Vacutainer® needle at the antecubital vein.

Different sampling tubes were employed: sodium citrate for coagulation/fibrinolysis factors and CRP; Sodium Fluoride/Potassium Oxalate for plasma Glucose; EDTA for lipids and fatty acids; Lithium Heparin for leptin and insulin.

Blood samples were ice-cooled immediately after collection. Plasma was separated by spinning samples during 10 minutes at 2500 RPM within one hour of collection and subsequently stored at -70°C until analysis within 8 weeks.

All samples for biochemical measures were measured by technicians blinded to status at the same time to avoid batch variation. Analyses were carried out at the laboratories of the Department of Pathological biochemistry, Clinical biochemistry and the Haemostasis, Thrombosis & Vascular Medicine section at the Glasgow Royal Infirmary.

Plasma leptin and Insulin

Plasma leptin was measured by an in-house radioimmunoassay (RIA) previously validated against the commercially available Linco assay (McConway *et al.*, 2000).

Insulin was measured by RIA (In vitro Diagnostic Coat-A-Count Insulin, Diagnostic Products Corp.).

Insulin sensitivity was assessed using the homeostasis model assessment using fasting plasma glucose and insulin concentrations as described by Mathews *et al.*, (1985).

HOMA-R = Insulin (mU/ml) · glucose (mmol/l)/22.5

Haemostatic factors

Samples for haemostatic factors were obtained in a sodium citrate vacutainer tube and delivered to the Haemostasis, Thrombosis & Vascular Medicine laboratory for analysis. ***Fibrinogen*** Clottable fibrinogen was measure using an automated Clauss method MDA180 (Organon Teknika, Cambridge, UK) with reagents from the manufacturer. The calibrant used was the 7th British Standard (NIBSC).

Factors FVIIc, VIII and IX were measured by standard clotting assays on an automated coagulometer (MDA 180,Organon Teknika, Cambridge, UK) using calibrants and reagents provided by the manufacturer.

C-reactive protein

C-reactive protein was measured immunologically using the BN ProSpec nephelometer (high sensitivity nephelometry, Dade Behring, Milton Keynes, UK) using calibrants and reagents provided by the manufacturer.

Lipids

Total and HDL-cholesterol and triglycerides in fasting blood were analysed at the department of Pathological Biochemistry, Glasgow Royal Infirmary, using commercial enzymatic colorimetric test kits (Roche diagnostics, Lewes, East Sussex, UK) in an automated Roche/Hitachi analyser 917. VLDL and LDL cholesterol were calculated using the Friedewald *et al.* (1972) equation:

LDL-cholesterol (mmol/l) =

Total-cholesterol (mmol/l– (HDL-cholesterol (mmol/l) + (triglycerides (mmol/l)/2.19)

VLDL-cholesterol =

Total cholesterol (mmol/l) – HDL cholesterol (mmol/l) – LDL cholesterol (mmol/l)

This recognised laboratory is CDC accredited for lipid measures and was responsible for lipid determination in major clinical trial such as the West of Scotland Coronary Prevention Study (WOSCOPS).

Adiponectin

Adiponectin was analysed using the Quantikine Adiponectin immunoassay, a 4.5-hour solid-phase ELISA employing the quantitative sandwich enzyme immunoassay technique

and designed to measure human Adiponectin in cell culture supernates, serum and plasma (R&D systems, Inc, UK).

Intercellular adhesion molecule (ICAM)

Plasma ICAM was analysed using the Human ICAM-1 immunoassay employing the quantitative sandwich enzyme immunoassay technique and designed to measure human Adiponectin in cell culture supernates, serum and plasma (R&D systems, Inc, UK).

Glucose

Plasma glucose was measured using an enzymatic in vitro test (Gluco-quant® Glucose/HK. Roche diagnostics, Mannheim, Germany) involving the phosphorylation of glucose with ATP by hexokinase to produce glucose-6-phosphate (G-6-P). The formation of G-6-P is directly proportional to the glucose concentration. This was measured using an automated clinical chemistry analyzer (IL2B 600).

Lipid sub-fractions

Apolipoprotein B (ApoB)-containing lipoproteins (VLDL-1, VLDL-2, IDL, and LDL) were isolated from plasma by a modification of the cumulative gradient ultracentrifugation procedure described by Lindgren *et al.* (1972). The TG, free cholesterol, cholesteryl ester, phospholipid, and protein content were assayed as previously described by Griffin *et al.* (1990).

The HDL sub-fraction distribution was determined using analytical ultracentrifugation (Beckman L-8 with optical scanning unit; Beckman High Wycombe, UK), which provides concentration values for HDL2 and HDL3 expressed as protein (Shepherd *et al.*, 1994).

Apo E Phenotype

There is evidence that ApoE polymorphisms may influence the blood lipid response to dietary interventions (Minihane *et al.*, 2000) as a possible confounding factor for the interpretation of results. Because no DNA sequence was performed, we assumed that ApoE phenotype represented ApoE genotype. ApoE phenotype was determined with the isoelectric focusing followed by Western blotting using an adaptation of the method described by Havekes *et al.* (1987).

Fatty acids

Fatty acids were analysed in plasma as their methyl esters (FAMEs) by gas chromatography-mass spectrometry (GC/MS) under electron impact ionisation as described by Montgomery *et al.*, (2003). Briefly, fatty acids were extracted via a modified Folch extraction (chloroform–methanol (1:1, v/v)) (Folch *et al.*, 1957) and derivatised with methanolic hydrochloric acid. An external standard of sixteen available non-esterified fatty acids of known concentration was derivatised concurrently with the samples. The external standard and the samples included an internal standard (15:0 triacylglycerol; Sigma-Aldrich, Poole, Dorset, UK) for calculation of response factors and concentrations. Fatty acid methyl esters were identified and quantified using gas chromatography-mass

spectrometry (GC/MS). The GC (Hewlett Packard 5890 Series II) was used in split mode (ratio 20:1). The carrier gas was helium (Grade A 99.996% pure, BOC, Surrey, U.K.) at a constant flow rate of 1 ml/min. The analytical column was a fused silica capillary column (BPX70, length 30 m, internal diameter 0.25 mm and film thickness 0.25 mm; SGE Europe Ltd.). The GC injector temperature was 250°C, and the injection volume 2 ml. The GC temperature programme was as follows: initial temperature 120°C for 2 min, increasing by 4°C min-1 to 180°C, then by 2°C min-1 to 194°C, followed by 30°C min-1 to 240°C, which was maintained for 1 min. The total GC run time for each sample is 26.5 min. The temperature of the transfer line between the GC and MS was 280°C. The MS (Hewlett Packard 5972) with electron impact ionisation was operated in scanning mode. EPA and DHA plasma levels were used in this study to determine compliance with fish consumption.

An estimation of CHD risk was performed using the PROCAM calculator (http://chdrisk.uni-muenster.de/calculator.php?iSprache=1&iVersion=1&iSiVersion=0)

5.2.3 Statistical analysis

A power calculation for the comparison of leptin was carried out. In the study by Winnicky *et al.* (2002) the standard deviation for leptin concentrations was 6.69 ng/ml for the fish eating group and 10.07 for the non-fish group. The mean difference between these groups was 6.8 ng/ml, i.e about one standard deviation. Based on this information, the sample size needed to detect such a difference, with a power of 90%, is 40 subjects. The coefficient of variation for serum leptin in our laboratory is < 10%. Therefore the proposed figure, i.e.

n=40, seems to be reasonable for this exploratory study and should in a crossover design be sufficient to detect a difference of 1 SD in serum leptin (6.8 ng/ml) attributable to fish eating. However we have increased the possible number of subjects to a maximum of 50 anticipating a 20% dropout rate (Petrie and Sabin, 2000).

Tests for normality were carried out for all data. Given the skewed distribution of several variables (adiponectin, insulin, leptin, ICAM-1, EPA, DHA, plasma lipids, CRP, fibrinogen) these were logarithmically transformed. Consumption of baseline and fish consumption data was obtained from 48 participants while data for the no-fish period was only available for 41 subjects. Paired Student t-test (baseline vs salmon, n=48; salmon vs no-fish, n=41) to compare continuous variables taking a significance level of 0.05. Pearson correlation coefficient was used to assess correlation between continuous variables. All statistical comparisons were carried out with SPSS 11.5 for windows (SPSS Inc., Chicago, Ill, USA).

5.3 RESULTS

Baseline data

The characteristics of the group under study are shown in **table 5.1**. On average the group was young; only two participants were above 40yrs of age. Normal weight subjects predominated with only one-fourth of the sample classified as overweight. Average BMI was 23.2 kg/m^2. The proportion of current drinkers was high, while a substantial proportion of the participants were never-smokers. All these factors reflect relatively high educational

levels. Distribution of subjects by ApoE phenotype is also shown. Eating behaviour was within normal ranges as indicated by dietary restraint, disinhibition and hunger scores of the three-factor eating questionnaire. Self-reported physical activity patterns showed a homogeneous high activity level in different areas (housework, work, and sports and exercise). Most of the group reported a discrete use of salt at the table.

Table 5.2 shows the frequency of weekly consumption of several food groups by the participants in this study. Fish was usually consumed once a week for each of white and oil-rich fish.

Baseline values for blood pressure **(table 5.4)** and clinical variables **(table 5.5)** fell within the normal range at all times as expected for healthy young non-obese people.

Salmon eating period and change from baseline (n=48; paired T-test)

Although baseline comparisons were not the main aim of the present study, the data have supportive value. The self-reported food and nutrient intake during the fish consumption intervention period is shown in **table 5.3.** The analysis of plasma fatty acids suggested good compliance with advice to eat salmon, as indicated by the increased concentrations of EPA and DHA above baseline (with two portions per week). A four-fold increase in EPA concentrations and two-fold increase in DHA was observed after four weeks of salmon consumption **(Table 5.4).**

Table 5.4 shows the changes in both sitting and standing blood pressure after the consumption of salmon. Values remained within the normal range at all times. Baseline body weight, waist circumference and percent body fat estimated by air displacement plethysmography remained stable during the study in keeping with maintenance of eating and physical activity levels. Dietary sodium, potassium, calcium and magnesium, factors known to influence blood pressure, were not different between treatment periods according to the self-reported food intake. Significantly lower blood pressure was observed in both sitting and upright positions after eating fish (on average -4 mmHg). These changes were large enough to produce significant decreases in mean arterial blood pressure (-2 to 3 mmHg).

The consumption of salmon significantly decreased fasting triglycerides by 13%, VLDL by 14%, together with significant increases of 8% in fasting levels of HDL cholesterol ($p<0.05$). Although no significant changes were observed in total and LDL cholesterol, the ratios of total cholesterol/HDL decreased significantly by 7% and the LDL/HDL by 9%. Adiponectin levels increased by 17% after salmon consumption ($P=0.018$) (**Table 5.5**).

These comparisons with baseline knowing that average fish consumption rose from 2 to 7 portions per week provides suggesting evidence for biological effects from salmon consumption and the potential "treatment effect" from effective health promotion advice.

Salmon vs no-fish period (n=41; paired T-test)

Comparison between the salmon and no-fish period provides the "treatment effect" if that is interpreted as the total effect of salmon eating.

Significantly ($P<0.01$) lower blood pressure was observed in both sitting and upright positions after eating fish (on average -5 mmHg). Diastolic blood pressure was also significantly lower on the fish intervention than after four weeks with no fish (-3 mmHg). The change produced by eating fish was a reduction of -4 mmHg in mean arterial blood pressure compared to the blood pressure after 4 weeks with no-fish.

The consumption of salmon significantly decreased fasting triglycerides by 15%, VLDL by 17% and LDL by 7%, together with significant increases of 5% in fasting levels of HDL cholesterol ($p<0.05$) (**Table 5.5**). The changes in the composition of lipoprotein sub-fractions are shown in tables **5.7**, **5.8** and **5.9**. The concentration of larger HDL-2 lipoprotein was significantly higher after salmon than with no-fish or baseline. No significant changes were seen in HDL-3 (**Table 5.7**). The concentration and composition of ApoB-containing lipoprotein are shown in **tables 5.8** and **5.9**. Significantly lower concentrations in IDL as well as in VLDL sub-fractions were observed with salmon consumption. An increase in the phospholipid content of these fractions was the only effects observed after salmon consumption.

Adiponectin was 8.39 μmol/l on salmon and 7.52 in no-fish (P=0.086) (**Table 5.5**). Values for most of the other "novel" inflammatory and haemostatic variables assessed in the present study were not significantly different between baseline and no-fish.

Results according to ApoE phenotype are shown in table **5.6**. Similar patterns of change in these variables were observed in both groups; however the small number of participants with allele 4 was too small for achieving statistical significance for most comparisons.

Plasma leptin levels correlated positively with BMI at all time points (Baseline r= 0.576, after salmon r=0.579, No-fish r=0.488; P< 0.001). Changes in body weight, body composition, plasma leptin and insulin concentrations during the study are shown in **table 5.5**. A neutral energy balance was observed during the study as indicated by no significant changes in body weight and percent body fat. No significant differences were observed either in plasma leptin, insulin, glucose or HOMA index, after consumption of salmon.

5.4 DISCUSSION

The consumption of fish is widely recommended for the prevention of CHD (WHO, 2003; Kris-Etherton *et al.*, 2002). Some of its benefits may reflect displacement of high saturated fat meat products; others may be related to components of fish itself. Fish oils appear to offer many of the benefits of fish itself (Connor, 2000) but this study contributes significantly to the scarce experimental data available and gives a real-life example in terms of reducing CHD risk factor levels with fish. For the present study salmon was chosen rather than other oil-rich fish such as sardines on the basis of pilot studies of its greater

acceptability. A four-week supplementation with daily salmon was the longest period acceptable among healthy volunteers. Dietary records indicated high compliance with daily salmon. No subject accepted the offer of earlier withdrawal if failing compliance was anticipated. In this event, compliance by unpaid volunteers was very good according to food intake records supported by rises in plasma EPA and DHA. The subjects were people who normally eat fish, but were prepared to go 4-weeks without fish. Among the Scottish population the consumption of fish at least twice a week is reported by 30% of men and women (SHS, 1998). The subjects usual oil-rich and white fish consumption however were 1 portion of each per week (range 1-3 total portions per week) thus achieving the current Scottish dietary target of 2 portions per week. Supporting these fish consumption data, baseline EPA and DHA values in the present study were 32% and 39% higher, respectively, than those recently reported for British adults who reported eating fish more than once a week (Rosell *et al.*, 2005). Eating a 125g portion of salmon each day for 4 weeks was therefore a major change. This level was not intended to represent an amount to be recommended and was probably unacceptable to 6 of the 48 subjects in whom there was no increase in plasma EPA or DHA.

5.4.1 Blood pressure

Recent epidemiological evidence suggests that consumption of meat, but not fish (or fruits and vegetables) was significantly associated with greater blood pressure over a 7-year period (Miura *et al.*, 2004). In addition, Pauletto *et al.* (1996) have reported significantly lower diastolic and systolic blood pressures with a fish-based diet compared to non-fish in Tanzanian subjects. Experimental studies show beneficial effects from fish oil supplements

particularly in hypertensive subjects (Morris *et al.*, 1993). There is very little experimental evidence on the use of fish itself, or from fish oil, in people with normal blood pressure (Morris *et al.*, 1993; Sacks *et al.*, 1994).

In the present study consumption of oil-rich fish provided, as 125 g salmon daily, to healthy subjects was effective in maintaining blood pressure below the levels found with no fish, and lower than their baseline values. These results were achieved in the absence of possible confounding factors such as changes in weight or in sodium intakes. Salmon consumption had a particularly marked effect on systolic blood pressure but diastolic blood pressure was still significantly lower than with no fish. The reductions in both systolic and diastolic blood pressure combined to produce significantly lower mean arterial blood pressure. Like all studies of dietary intervention in a real-life or "ecological" setting, the results of the present study depend on self-reported dietary compliance and it is of course not possible to have a "placebo" which resembles salmon but is not. Since other foods were consumed instead of fish during the control period, it is possible that they had a specific blood pressure elevating effects, rather than salmon having a blood pressure lowering action. However, the alternative foods in this control arm were self-selected from options including cheddar cheese, pasta, chicken, and turkey. These are not foods known to increase blood pressure.

It is possible that the recording period was insufficient to characterise dietary sodium, potassium, calcium and magnesium, nutrients known to influence blood pressure. However, given that the participants in this study were healthy subjects and the recording was done

during non-consecutive days to decrease variability, it seems reasonable to assume that indeed the consumption of these nutrients was similar in both treatment periods.

The subjects who volunteered for this study enjoyed fish and ate it 1 to 3 times per week as part of their habitual pre-study diets. Blood pressure at baseline was higher than that after salmon 7 times a week but lower than levels with no-fish, so a more realistic consumption of 3 or 4 portions weekly may be sufficient to maintain a lower blood pressure. If an order effect were to operate, one might expect blood pressure to have been lower, not higher, at the end of this study. Regression towards the mean cannot explain these effects since subjects were not selected on the basis of their blood pressure. The relevance of blood pressure as a risk factor for CHD has been shown at early ages; diastolic hypertension imposes a stronger risk before 50 years of age while systolic hypertension does it at older ages (Franklin *et al.*, 2001). It has been shown that modest decreases in systolic blood pressure such those observed in the present study (e.g. ~5 mmHg) may have a substantial impact at the population level resulting in a reductions of 14% in mortality due to stroke, 9% in mortality due to CHD, and 7% in all-cause mortality (Chobanian *et al.*, 2003).

Earlier smaller studies, assessing the consumption of fish during two-week periods, also reported significant changes in systolic and diastolic blood pressure in healthy volunteers after consumption of mackerel but not herring (Singer *et al.*, 1983). The same group reported similar results in patients with type IV and V hyperlipoproteinaemia (Singer *et al.*, 1985a) and changes in systolic blood pressure in subjects with mild hypertension (Singer *et al.*, 1985b). In a third study in mildly hypertensive subjects, these authors (Singer *et al.*, 1986) reported significantly lower blood pressure after 8 months of consumption of

mackerel 3 times per week similarly to the present study; these values rose to basal levels after two months of a normal diet. Only one study (von Houwelingen *et al.*, 1987) reported no significant difference in blood pressure changes in healthy volunteers after eating 100 g/d of either mackerel or meat during six weeks. These authors (von Houwelingen *et al.*, 1987) reported a decrease in the compliance with time, particularly when eating mackerel.

In conclusion, the present findings support the consumption of salmon as a means to lowering blood pressure. Daily consumption of salmon or fish in general is usual in many parts of the world; however the results of this study together with the available literature do not necessarily justify recommending daily consumption, or even a greater intake than the current recommendation of one to two servings per week (WHO, 2003). However in many populations –eg northern Europe fish is rarely eaten. Larger and longer, studies would be necessary to establish "dose-responses" and to identify any threshold levels to define an optimal intake.

5.4.2 Lipids

The lowering effect of fish consumption and fish oils on the levels of triglycerides (Nordoy *et al.*, 1993; Harris *et al.*, 1988) and VLDL (Harris *et al.*, 1990) is well documented. Changes in total cholesterol and other cholesterol sub-fractions might be more evident in hypercholesterolemic subjects. A recent study suggests that a low fat diet decreases cholesterol in hypercholesterolemic subjects regardless of the type of protein consumed: lean-fish, lean-beef, or poultry (Beauchesne-Rondeau *et al.*, 2003).

In the present study, in normolipemic subjects, significant decreases in triglycerides and VLDL and LDL cholesterol together with an increase in HDL cholesterol were found with 4-weeks consumption of salmon. These changes were of similar magnitude to those reported by Beauchesne-Rondeau *et al.* (2003) in hypercholesterolemic patients after 26-days of a complex lipid-lowering diet based on the American Heart Association recommendations.

Decreases in VLDL comprised significant reduction in both main particles classes: large, buoyant VLDL-1 (Svedberg flotation rate 60 to 400) and small, dense VLDL-2 particles (Svedberg flotation rate 20 to 60). Among the mechanisms behind the lowering effects of n-3 fatty acids on triglycerides and VLDL is a decreased hepatic output as a result of both an increase in fatty acid oxidation and a decrease in lipogenesis (Jump *et al.*, 1997 Hwang 2000). A reduced VLDL and chylomicrons synthesis seems likely given that there are no associations of reduced fat absorption with consumption of fish oils and the diet of the participants remained unchanged. Since EPA and DHA have ligand binding activity for peroxisome proliferator-activated receptors (PPAR) that regulate genes involved in lipid and carbohydrate metabolism as well as the inflammatory response, a mechanism involving PPAR may be in part responsible for these effects (Hwang, 2000).

In the present study, consumption of salmon did not increase LDL cholesterol as has been previously shown with fish oils (Connor, 2000), and explained as a lack of effect of fish oils on insulin sensitivity (Rivellese *et al.*, 1996; Patti *et al.*, 1999). An increase in LDL during fish oil supplementation has been suggested as a positive sign (Suzakawa et al., 1995). A reduction in plasma triglyceride would be expected to reduce the triglyceride

content of LDL leading to an increase in LDL particles size, which are less susceptible to oxidation than small dense LDL particles, and therefore might be expected to reduce the atherogenic risk.

In the present study, a lower concentration of LDL cholesterol was observed with fish consumption compared with no-fish as well as baseline values; only the difference with no-fish was statistically significant. No significant effect of fish consumption on insulin sensitivity, as estimated by the HOMA-IR, was observed but the subjects all had normal insulin sensitivity and LDL concentrations.

Results from food records indicated no significant changes in saturated fat intake to which could also explain the lower LDL cholesterol levels. Despite the great emphasis placed in maintaining eating patterns during the study and the fact that participants were not known dieters and successfully maintained a stable body weight and body fat during the study, it is important to keep in mind that accuracy and veracity of dietary reports is a common problem.

It is also important to keep in mind that LDL-cholesterol is not measured directly but calculated. No significant changes were observed in LDL-mass or composition, as determined by centrifugation.

The lowering effect on triglycerides observed in the present study has been associated with increases in HDL-cholesterol. The mechanisms behind this process involve less triglyceride being transferred to HDL from triglyceride-rich lipoproteins. Triglyceride-enrichment of

HDL may be an important factor in lowering HDL-cholesterol particularly in hypertriglyceridaemic subjects. Triglicride-enrichment results in smaller HDL particles, intrinsically unstable in the circulation, that are catabolised at a faster rate leading to a reduced HDL-cholesterol levels and particles (Lamarche et al., 1999).

There is evidence that fish consumption may increase the levels of HDL cholesterol not only through mechanism involving n-3 fatty acids but also possibly protein (Bergeron *et al.*, 1992). The higher values in HDL cholesterol in the present study were due to increases in the HDL-2 sub-fraction, the cardio-protective sub-fraction of HDL, but not in HDL-3. No significant changes however, were observed in total cholesterol levels in the present study.

The improvements in triglycerides, LDL and HDL, all diagnostic criteria for metabolic syndrome, suggest improved insulin sensitivity (Patti *et al.*, 1999) and this mechanism could explain a lower factor VII if there was one.

Taken as a whole, the combination of blood pressure and lipid improvements seen with salmon intake, if sustained over time, would equate to around a 25% risk reduction for CHD events based on the PROCAM risk calculator (http://chdrisk.uni-muenster.de/calculator.php?iSprache=1&iVersion=1&iSiVersion=0), an effect approaching the level of benefit of drug interventions for prevention of CHD with statins and ACE inhibitors (Yusuf *et al.*, 2000) or indeed fibrates (Rubbins *et al.*, 1999).

5.4.3 Leptin

High levels of leptin are common in human obesity and evidence from animal and human studies indicates a relationship with metabolic syndrome through different mechanisms such as vascular function, by presenting angiogenic activity or increasing oxidative stress in endothelial cells, as well as possibly contributing to an increased arterial pressure (Wallace *et al.*, 2001; Unger and Orci, 2002; Singhal *et al.*, 2002). A recent study (Winniky *et al.*, 2002) in Tanzanian villagers, reported an association between daily consumption of fish (300 to 600 g) and lower serum leptin. Fish-eating men and women had lower levels of leptin than non-fish eaters. Fish-eating women had lower leptin levels than non-fish-eating men after adjusting for confounding factors; a surprising finding given that leptin levels in women are normally higher than in men. In addition, Mori et al. (2004) have shown that fish consumption, as part of a weight-reducing regimen, was more effective than either measure alone at reducing leptin levels.

Increases in leptin concentration were associated with high saturated fat diets in mice, while a high n-3 PUFA fish-oil diet decreased leptin and corrected hyperleptinaemia after saturated fat diet (Wang *et al.*, 2002). In humans, rapeseed oil showed increases of plasma leptin in men and decreases in women (Kratz *et al.*, 2002); however this study lacked control for menstrual cycle effects, which confound the interpretation of these results.

The present experimental study in healthy volunteers showed no significant acute changes in leptin after the consumption of salmon. These healthy non-obese subjects were heavier than the Tanzanian subjects as indicated by mean BMI (23.2 vs 20.3 kg/m^2) and a higher

proportion of overweight subjects (25% vs <5%). The levels of leptin observed in the present study resembled those for non fish-eating women and fish-eating men. In this study, factors known to affect leptin levels such as weight changes and phase of the menstrual cycle in women were well controlled. A neutral energy balance observed during this study reflected maintenance of eating and physical activity patterns; factors difficult to control in epidemiological studies. However it is still possible that the differences reported by Winniky *et al.* (2002) might be related to the chronic consumption of fish by these groups. Unfortunately no other study assessing the relationship of leptin with fish consumption is available in the literature.

5.4.4 Adiponectin

No other studies have assessed the effects of fish consumption on adiponectin in humans. Recent evidence in mice shows that fish oil down-regulates the lowering effects of conjugated linoleic acid on leptin and adiponectin (Ide, 2005). Plasma adiponectin levels are low in obese animals and humans and particularly in patients with type-2 diabetes mellitus and metabolic syndrome (Trujillo Scherer, 2005). It promotes fatty acid oxidation, decreases plasma triglycerides, and increases insulin sensitivity (Yamauchi *et al.*, 2001; Fruebis *et al.*, 2001). Adiponectin may reduce the inflammatory response and possibly atherogenesis by suppressing migration of monocytes/macrophages and their transformation into foam cells. It is notable that PPAR agonists substantially increase adiponectin. A recent study by Rossi et al. (2005) showed that consumption of fish oil, for seven months, positively regulates plasma leptin and adiponectin levels in sucrose-fed, insulin-resistant rats.

In the present study we observed a significant increase in adiponectin levels, as compared to baseline, after consumption of salmon. This increase was independent of BMI and age as well as of other confounding factors such as weight changes. Fish consumption before baseline was not controlled but dietary records suggest a moderate consumption and plasma EPA and DHA at baseline were similar to the levels after 4-weeks with no-fish. Baseline EPA and DHA values in the present study were 32 and 39% higher than those recently reported for British adults reporting eating fish more than once a week (Rosell *et al.*, 2005). The difference in the adiponectin concentration in the controlled salmon and no-fish periods achieved a p-value of 0.086 for all subjects, and P=0.50 for those with ApoE phenotype 3/2 and 3/3 (n=30). These data are thus suggestive of an effect of salmon consumption to increase adiponectin and this increase insulin sensitivity and the related metabolic variables. The effect seen in these healthy subjects was small. It will be of great interest to evaluate this effect in people with diabetes or metabolic syndrome.

These data support the existence of a mechanism involving adiponectin and fish oils, on the assumption that fish oils are the active component of salmon in lipid metabolism. No significant changes were observed in other variables theoretically modulated also by adiponectin, such as ICAM.

Among the possible mechanisms involved on the regulation of adiponectin by EPA and DHA is the activation of PPARα. PPARα expressed mainly in skeletal muscle, heart, liver, and kidney and thought to regulate many genes involved in the beta-oxidation of fatty acids. Induction of peroxisomes by this mechanism leads to a reduction in blood triglyceride levels (Jump *et al.*, 1997). However modulation of interleukins has been shown

to be independent of PPAR mechanism (Zhang and Fritsche, 2004). Therefore, there is a possibility that other nutritional components of fish may have an impact on lipid metabolism.

5.4.5 Insulin sensitivity

The present study included healthy subjects not known to be at risk of diabetes or metabolic syndrome therefore HOMA indices (range 0.49, 2.75) were well below the cut-offs used to indicate insulin resistance (>3.0). No significant differences in insulin sensitivity were seen between treatments, suggesting that improvements in metabolic syndrome features from salmon consumption might not entirely be modulated by a change in insulin sensitivity.

5.4.6 Haemostatic factors

A range of haemostatic variables was measured, however no significant differences were found after fish supplementation. These results are consistent with recent findings in the literature. A recent study (Finnegan *et al.*, 2003) in moderately hyperlipidemic subjects showed no effect on any fibrinolytic factor (factors VIIa, VIIc, VIIag, XIIa, XIIag, fibrinogen concentrations, plasminogen activator inhibitor-1 or tissue plasminogen activator activity) after 6-months supplementation with 0.8 or 1.7g/d EPA+DHA, 4.5 or 9.5g/d α-linolenic acid or control (linoleic acid) provided as oil supplements. Similar results have been found in untreated hypertensive subjects (Toft *et al.*, 1997).

5.4.7 Genetic determinants of responses

It was beyond the scope of the present study to investigate genetics in response to dietary change but awareness of the issues led to measure Apo E phenotype. There was no definite indication for an influence in the small numbers with different phenotype.

5.4.8 Exclusion of non-compliant subjects

The mean data on this chapter were presented from intent-to-treat analysis however compliance is never 100% and non-compliant subjects can lead to bias from underestimation of biological effects. In the present study, DHA and EPA were found to rise substantially in 42 of the 48 subjects who completed the salmon consumption period. In 6 subjects there was no rise. **Table 5.10** shows the salmon – no-fish differences in the key outcome measures potentially modified by salmon for these two subgroups. In the non-compliant subjects there were no changes in BP while triglycerides and VLDL increased significantly during the no-fish period.

These results suggest that EPA and DHA were appropriate tests as biomarkers for compliance. Excluding these 6 non-compliers did not alter the main results.

5.4.9 Possible adverse effects from high fish consumption

Consumption of oil-rich fish or fish oil, containing significant amounts of long-chain n-3 PUFAs, may result in the enrichment of LDL with molecules that are highly susceptible to

lipid peroxidation. Peroxidation of LDL is considered a risk factor for atherogenesis with its known deleterious consequences (Kita et al., 2001). However, several recent studies in hyperlipidemic and healthy subjects report no significant effects of different amounts of omega-3 fatty acids (from 1 to 5 g/d) on LDL oxidation (Brude et al., 1997, Higgins et al., 2001) The amount of n-3 fatty acids contained in 125 g of salmon consumed in the present study (2.5 g) are within these ranges. In the present study LDL concentrations did not change and oxidised LDL was not measured.

A number of theoretical hazards of high fish consumption have been raised including intake of heavy metals and dioxins, which could not be addressed.

Prolonged bleeding time has been reported, but was not observed in the present study. Bleeding time was not measured directly but there was no significant changes in coagulation factor VII or other haemostatic variables.

5.4.10 Conclusion

In conclusion, consuming a diet enriched with salmon daily for four weeks improved traditional risk factors (lipids and blood pressure) and which predict CHD risk reduction of around 25% using PROCAM (**Figure 1**). It decreased systolic blood pressure, LDL, triglycerides, and VLDL, and increased HDL cholesterol. It appears possible that the mechanism of these effects involves increased adiponectin, which correlated with changes in triglycerides and VLDL but in the healthy, young subjects no increase in insulin sensitivity or related change in factor VII was detected. The results of this study together

with the available literature do not necessarily justify recommending daily consumption, or even a greater intake than the current recommendation of 2 servings/week. Nevertheless our results help support the case for increased fish intake generally and for further larger studies in subjects at elevated risk for cardiovascular disease.

5.5 New research questions

This chapter has provided some experimental data on the effects of fish consumption on indices of the metabolic syndrome.

1. Does fish improve metabolic indices in patients with type-2 diabetes and metabolic syndrome?

2. How much fish is necessary to achieve a clinically valuable effect? Are 3 or 2 portions per week sufficient?

3. Are the effects of fish consumption entirely attributable to fish oil?

4. Are the effects of fish consumption mediated by adiponectin and if so is insulin action involved? And is ApoE, or other, phenotype important?

Table 5.1 Baseline characteristics in 48 subjects

	Mean ± SD
Overweight (BMI 25-29.9) (%)	25
Women (%)	66.6
Age (yrs)	28.2 ± 7.8
Height (m)	1.68 ± 0.09
Weight (kg)	65.4 ± 10.4
Waist (cm)	75.6 ± 7.4
Hip (cm)	96.6 ± 7.6
BMI (kg/m2)	23.2 ± 3.1
ApoE phenotype	
3/2 n (%)	5 (10.4)
3/3 n (%)	31 (64.6)
4/3 n (%)	10 (20.8)
4/4 n (%)	2 (4.2)
Eating behaviour scores	
Dietary restraint	7.2 ± 4.2
Disinhibition	5.3 ± 3.1
Perceived hunger	4.6 ± 2.3
Smoking habits (%)	
Current /Former/Never smoker	20 /8 /66
Current drinkers (%)	84
Use of salt at the table	
Generally add salt without tasting first (%)	6
Taste food and then generally add salt (%)	9
Taste food but occasionally add salt (%)	32
Rarely or never add salt at table (%)	53

Table 5.2 Frequency of food consumed before the study

	Mean	SD
Per day		
Cereals	4.1	1.9
Fruits	1.7	1.3
Cooked green vegetables	0.8	0.7
Cooked root vegetables	0.7	0.6
Salad	0.8	0.7
Chips	0.2	0.3
Potatoes, pasta, rice	1.1	0.9
Poultry	0.4	0.2
Meat products	0.4	0.4
Meat	0.3	0.2
Cheese	0.8	0.6
Beans, pulses	0.3	0.3
Sweets	0.8	0.8
Ice cream	0.1	0.2
Crisps	0.6	0.9
Fruit juice	0.7	0.6
Soft drinks	0.8	1.0
Cakes	0.4	0.5
Biscuits	0.8	0.8
Eggs	0.3	0.3
Per week		
Oil-rich fish	1.1	0.5
White fish	0.9	0.4

Table 5.3 Self-reported daily nutrient intakes in 48 healthy adult subjects during the fish consumption period

	All subjects (n= 48)	Men (n= 16)	Women (n= 32)
Energy (Kilojoules)	7938.7 ± 2298.0	9554.8 ± 3084.0	6678.8 ± 1880.4
(Kilocalories)	1888.7 ± 546.3	2388.7 ± 746.2	1669.7 ± 445.1
Carbohydrates (%)	46.2 ± 8.2	45.8 ± 7.9	47.1 ± 8.9
Protein (%)	17.9 ± 5.3	17.9 ± 6.2	15.8 ± 4.9
Fat (%)	34.5 ± 7.0	36.2 ± 8.6	33.4 ± 6.5
Fatty acids			
Poly-unsaturated (%)	6.1 ± 1.9	7.2 ± 3.4	6.0 ± 1.8
Mono-unsaturated (%)	12.2 ± 3.3	13.5 ± 3.9	12.0 ± 3.0
Saturated (%)	11.1 ± 2.7	12.1 ± 3.5	10.8 ± 2.5
Alcohol (%)	4.3 ± 4.5	7.3 ± 3.6	3.8 ± 4.9

Lipid and fatty acid content of 125 g salmon

(Eaten daily within the diet above)

Total lipids (g)	13.56
Total n-3 fatty acids (g)	2.51
DHA (g)	1.62
EPA (g)	0.77
Total n-6 (g)	2.23
Ratio n3/n6	1.13

Table 5.4 Body weight and blood pressure and fatty acids during the study

	Baseline n=48	**After salmon n=48**	**Mean difference (95 CI) baseline - salmon**	**P**	**No fish n=41**	**Mean difference (95 CI) salmon - no-fish**	**P**
Body weight (kg)	65.5 ± 10.4	65.6 ± 10.4	-0.13 (-0.3, 0.1)	0.866	65.7 ± 10.5	-0.21 ()-0.25, 0.20	.750
Waist (cm)	75.6 ± 7.4	75.5 ± 7.7	-0.14 (-0.1, 0.5)	0.888	75.6 ± 7.5	-0.3 (-0.5, 0.30)	.800
Body fat (%)	20.1 ± 8.5	20.0 ± 8.5	0.1 (-0.2, 0.3)	0.879	20.1 ± 8.9	0.0 (-0.1, 0.1)	.950
Blood Pressure (mmHg)							
Sitting systolic	113.5 ± 10.7	109.8 ± 9.8	3.69 (1.30, 5.96)	0.003	114.4 ± 8.6	-4.6 (-7.1, -2.0)	0.001
Sitting diastolic	72.7 ± 7.6	71.3 ± 6.5	1.4 (-0.5, 3.2)	0.144	74.6 ± 8.1	-2.9 (-5.0, -0.9)	0.009
Sitting pulse (beats/min)	72.7 ± 10.8	70.4 ± 10.4	2.3 (-0.9, 5.4)	0.162	73.6 ± 10.9	-2.8 (-5.6, 0.1)	0.066
Standing systolic	115.5 ± 10.0	111.3 ± 10.8	4.2 (1.5, 6.9)	0.003	116.6 ± 10.3	-5.5 (-8.1, -2.8)	0.00002
Standing diastolic	77.2 ± 6.6	76.2 ± 6.7	1.1 (-0.8, 2.9)	0.258	78.6 ± 7.6	-2.0 (-4.1, 0.1)	0.078
Standing pulse (beats/min)	78.8 ± 10.5	77.8 ± 10.3	0.8 (-2.3, 3.9)	0.619	82.9 ± 11.6	-3.9 (-6.8, -0.9)	0.011
Mean arterial BP sitting	86.3 ± 7.5	84.2 ± 6.6	2.1 (0.2, 3.9)	0.027	87.7 ± 7.3	-3.5 (-5.5, -1.6)	0.001
Mean arterial BP standing	89.9 ± 7.3	87.1 ± 8.5	2.9 (0.6, 5.1)	0.015	91.2 ± 6.9	-4.0 (-6.5, -1.5)	0.003
Plasma Fatty acids							
EPA (μmol/L) (n=43)	30 ± 16	131 ± 55	-101 (-114, -79)	0.0001^{-14}	31 ± 27	98 (114, 75)	0.00009^{-9}
DHA (μmol/L) (n=43)	75 ± 31	152 ± 50	-77 (-89, -61)	0.0001^{-13}	78 ± 33	79 (64, 94)	0.00003^{-9}

Values are provided as mean ± SD as well as the paired differences (mean difference with 95% CI)
P-values are for paired t-tests (baseline vs salmon n=48; salmon vs no-fish n=41)
MABP=Mean arterial blood pressure = [(diastolic BP x 2) + systolic BP]/3

Table 5.5 Metabolic after consumption of fish

Characteristic	Baseline n=48	After salmon N=48	Mean difference (95 CI) baseline - salmon		No-fish n=41	Mean difference (95 CI) salmon - no-fish	P
Plasma leptin (ng/ml)	9.8 ± 2.8	10.2 ± 2.6	-0.4 (-0.85, 1.05)	0.304	10.6 ± 2.7	-0.4 (-1.9, 0.7)	0.327
Plasma insulin (μU/ml)	6.5 ± 1.5	6.9 ± 1.8	-0.4 (-1,7, 0.4)	0.864	7.6 ± 1.6	-0.7 (-1.7, 0.7)	0.631
Glucose (mmol/l)	4.78 ± 0.32	4.83 ± 0.38	0.05 (-0.15, 0.05)	0.289	4.81 ± 0.36	0.01 (-0.10, 0.13)	0.802
HOMA ratio	1.49 ± 0.36	1.64 ± 0.87	-0.16 (-0.39, 0.07)	0.177	1.78 ± 0.91	-0.16 (-0.43, 0.11)	0.232
Triacyglycerol (mmol/l)	0.94 ± 0.33	0.82 ± 0.35	0.12 (0.05, 0.20)	0.002	0.97 ± 0.37	-0.13 (-0.24, -0.1)	0.017
Total cholesterol (mmol/l)	4.32 ± 0.76	4.33 ± 0.85	-0.01 (-0.18, 0.16)	0.894	4.37 ± 0.71	-0.10 (-0.29, 0.01)	0.213
VLDL cholesterol (mmol/l)	0.43 ± 0.15	0.37 ± 0.16	0.06 (0.02, 0.09)	0.003	0.44 ± 0.17	-0.07 (-0.11, -0.01)	0.016
LDL cholesterol (mmol/l)	2.39 ± 0.69	2.31 ± 0.74	0.08 (-0.09, 0.23)	0.530	2.48 ± 0.64	-0.17 (-0.26, 0.00)	0.032
HDL cholesterol (mmol/l)	1.31 ± 0.30	1.42 ± 0.34	0.11 (-0.16, -0.05)	0.001	1.33 ± 0.28	0.09 (0.01, 0.15)	0.040
Total chol/HDL ratio	3.24 ± 0.86	3.01 ± 0.88	0.23 (0.12, 0.32)	0.00005	3.30 ± 0.78	-0.25 (-0.38, -0.11)	0.001
LDL/HDL ratio	1.91 ± 0.74	1.74 ± 0.74	0.17 (0.07, 0.26)	0.001	1.97 ± 0.71	-0.20 (-0.29, -0.09)	0.0004
Non-HDL cholesterol	2.94 ± 0.71	2.84 ± 0.80	0.10 (-0.04, 0.23)	0.168	3.03 ± 1.25	-0.18 (-0.33, -0.03)	0.029

Values are provided as mean ± SD as well as the paired differences (mean difference with 95% CI)
P-values are for paired t-tests (baseline vs salmon n=48; salmon vs no-fish n=41)

Table 5.5 Continued...

ICAM (ng/ml)	215.4 ± 42.5	210.4 ± 49.9	5.0 (6.21, -7.50)	0.425	216.1 ± 44.2	-3.1 (-13.65, 7.41)	0.500
Adiponectin ((ug/ml)/1000)	7.18 ± 3.99	8.39 ± 4.33	1.21 (0.75, 1.67)	0.018	7.52 ± 4.10	0.57 (-0.41, 0.01)	0.086
Fibrinogen (g/l)	2.8 ± 0.6	2.9 ± 0.5	-0.1 (0.0, 0.29)	0.879	2.9 ± 0.6	0.01 (-0.11, 0.10)	0.979
FVIIc (iu/dl)	129.5 ± 29.1	134.0 ± 27.8	-4.5 (-11.0, 2.3)	0.197	133.8 ± 26.2	0.20 (-7.89, 8.37)	0.640
FVIII (iu/dl)	122.3 ± 28.9	120.6 ± 26.6	1.7 (-5.4, 8.3)	0.677	121.1 ± 27.9	-0.41 (-7.6, 6.8)	0.973
FIX (iu/dl)	120.3 ± 19.3	121.3 ± 15.9	-1.0 (-4.4, 5.9)	0.354	121.1 ± 16.7	0.20 (-4.36, 4.79)	0.931
CRP (mg/l)	0.46 ± 0.42	0.51 ± 0.43	-0.05 (-0.11, 0.17)	0.247	0.53 ± 0.50	-0.02 (-0.11, 0.09)	0.864

Values are provided as mean ± SD as well as the paired differences (mean difference with 95% CI)
P-values are for paired t-tests (baseline vs salmon n=48; salmon vs no-fish n=41)

Table 5.6 Metabolic changes after consumption of fish

Characteristic	ApoE phenotype allele 3				ApoE phenotype allele 4			
	Salmon n=30	No-fish n=30	Mean difference (95%CI)	P	Salmon n=11	No-fish n=11	Mean difference (95%CI)	P
Plasma leptin (ng/ml)	12.94 ± 2.49	13.09 ± 2.62	-0.15 (-0.37, 0.00)	.908	14.65 ± 3.25	15.30 ± 3.30	-0.65 (-1.05, 0.49)	.136
Plasma insulin (μU/ml)	8.23 ± 1.88	7.81 ± 1.65	0.42 (-0.75, 2.28)	.681	6.84 ± 2.06	5.97 ± 2.10	0.87 (0.83, 0.92)	.701
Glucose (mmol/l)	4.82 ± 0.38	4.75 ± 0.35	-0.07 (-0.07, 0.20)	.310	4.83 ± 0.36	5.01 ± 0.34	-0.07 (-0.07, 0.20)	.104
HOMA ratio	1.68 ± 0.97	1.90 ± 0.90	-0.23 (-0.57, 0.12)	.188	1.41 ± 0.98	1.34 ± 0.44	-0.07 (-0.13, 0.26)	.445
Triacyglycerol (mmol/l)	0.76 ± 0.36	0.89 ± 0.37	-0.13 (-0.25, 0.01)	.010	0.82 ± 0.38	0.97 ± 0.39	-0.15 (-0.44, 015)	.195
Total cholesterol (mmol/l)	4.14 ± 0.77	4.30 ± 0.77	-0.16 (-0.26, 0.10)	.179	4.31 ± 0.65	4.37 ± 0.55	-0.06 (-0.42, 0.31)	.694
VLDL cholesterol (mmol/l)	0.34 ± 0.16	0.41 ± 0.16	-0.06 (-0.11, 0.00)	.008	0.38 ± 0.17	0.44 ± 0.18	-0.06 (-0.20, 0.07)	.197
LDL cholesterol (mmol/l)	2.28 ± 0.83	2.47 ± 0.70	-0.12 (-0.28, 0.04)	.043	2.39 ± 0.45	2.53 ± 0.41	-0.14 (-0.39, 0.13)	.252
HDL cholesterol (mmol/l)	1.43 ± 0.28	1.37 ± 0.27	0.06 (-0.04, 0.15)	.193	1.48 ± 0.38	1.35 ± 0.32	0.14 (0.06, 0.21)	.002
Total chol/HDL ratio	3.07 ± 0.97	3.28 ± 0.79	-0.21 (-0.34, -0.08)	.003	3.06 ± 0.85	3.37 ± 0.79	-0.32 (-0.71, 0.07)	.099
LDL/HDL ratio	1.79 ± 0.83	1.95 ± 0.73	-0.17 (0.28, -0.06)	.003	1.75 ± 0.67	2.00 ± 0.66	-0.26 (-0.53, 0.01)	.061

Values are provided as mean ± SD as well as the paired differences (mean difference with 95% CI)
P-values are for paired t-tests

Table 5.6 Continued...

Characteristic	ApoE phenotype allele 3				ApoE phenotype allele 4			
	Salmon n=30	No-fish n=30	Mean difference (95%CI)	P	Salmon n=11	No-fish n=11	Mean difference (95%CI)	P
Non-HDL cholesterol	2.65 ± 0.94	2.90 ± 0.74	-0.25, (-0.45, -0.01)	.011	2.78 ± 0.58	2.98 ± 0.53	-0.20 (-0.56, 0.18)	.204
ICAM (ng/ml)	208.5 ± 52.3	210.7 ± 49.7	-1.7 (-14.5, 11.0)	.673	206.6 ± 30.4	214.0 ± 26.3	-7.4 (-28.8, 15.2)	.500
Adiponectin ((ug/ml)/1000)	8.18 ± 1.80	7.15 ± 1.82	1.03 (0.89, 1.32)	.050	6.73 ± 1.89	7.46 ± 1.90	-0.73 (-1.18, 0.28)	.406
Fibrinogen (g/l)	2.89 ± 0.47	2.89 ± 0.53	-0.00 (-5.2, 13.20)	.995	2.97 ± 0.57	2.99 ± 0.77	-0.3 (-0.30, 0.25)	.833
FVIIc (iu/dl)	136.2 ± 28.5	132.2 ± 27.3	4.0 (-5.2, 13.20)	.378	128.2 ± 26.6	138.0 ± 22.5	-9.8 (-28.4, 8.8)	.260
FVIII (iu/dl)	122.00 ± 25.4	120.4 ± 25.2	1.54 (-6.89, 9.97)	.709	117.1 ± 30.9	122.78 ±	-5.7 (-22.1, 10.8)	.450
FIX (iu/dl)	120.4 ± 15.8	122.7 ± 15.8	-2.29 (-7.95, 3.37)	.411	123.6± 17.1	116.7 ± 18.9	6.88 (-0.04, 13.82)	.051
CRP (mg/l)	0.42 ± 0.43	0.47 ± 0.42	-0.05 (-0.07, 0.04)	.460	0.88 ± 0.85	0.66 ± 0.75	0.22 (-0.11, 0.32)	.372

Values are provided as mean ± SD as well as the paired differences (mean difference with 95% CI)
P-values are for paired t-tests

Table 5.7 Concentration and composition of HDL sub-fractions

	Baseline n=48	Salmon n=48	Mean difference (95 CI) baseline - salmon	p	No-fish n=41	Mean Difference (95% CI) Salmon - no-fish	P
HDL2 Mass (mg/dl)	151.66 ± 54.42	175.43 ± 64.00	-23.77 (-38.70, -8.84)	0.002	149.14 ± 58.06	18.28 (4.79, 31.76)	0.009
Composition:							
Protein (%)	21.75 ± 2.03	22.63 ± 2.25	-0.88 (-1.57, -0.19)	0.014	21.53 ± 2.23	1.02 (0.06, 1.97)	0.038
Free Cholesterol (%)	3.83 ± 0.76	4.29 ± 0.64	-0.46 (-0.72, -0.20)	0.001	4.01 ± 0.75	0.24 (-0.01, 0.49)	0.057
Cholesteryl ester (%)	27.00 ± 1.98	27.28 ± 1.88	-0.28 (-0.90, 0.35)	0.377	27.42 ± 2.21	-0.10 (-0.81, 0.60)	0.773
Trigliceride (%)	43.94 ± 2.99	42.78 ± 3.00	1.16 (0.08, 2.24)	0.036	43.67 ± 2.48	-0.88 (-1.99, 0.24)	0.119
Phospholipidid (%)	3.48 ± 1.04	3.03 ± 1.12	0.45 (0.14, 0.77)	0.006	3.37 ± 1.35	-0.28 (-0.67, 0.11)	0.157
HDL-3 Mass (mg/dl)	90.43 ± 23.24	85.85 ± 20.60	4.58 (-3.38, 12.53)	0.253	88.18 ± 25.36	-2.85 (-12.71, 7.02)	0.563
Composition:							
Protein (%)	14.16 ± 2.45	14.84 ± 2.70	-0.68 (-1.78, 0.42)	0.221	14.26 ± 2.10	0.34 (-0.74, 1.42)	0.527
Free Cholesterol (%)	2.63 ± 0.82	2.72 ± 0.72	-0.09 (-0.37, 0.18)	0.490	2.48 ± 0.55	0.31 (0.05, 0.58)	0.022
Cholesteryl ester (%)	18.66 ± 2.12	18.51 ± 2.04	0.15 (-0.52, 0.82)	0.651	19.24 ± 2.33	-0.66 (-1.50, 0.19)	0.123
Trigliceride (%)	62.44 ± 3.68	61.81 ± 3.20	0.64 (-0.67, 1.95)	0.330	61.90 ± 3.60	-0.07 (-1.53, 1.39)	0.920
Phospholipidid (%)	2.11 ± 0.73	2.12 ± 1.88	-0.02 (-0.59, 0.55)	0.953	2.12 ± 1.36	0.08 (-0.63, 0.78)	0.825

Values are provided as mean ± SD as well as the paired differences (mean difference with 95% CI)
P-values are for paired t-tests (baseline vs salmon n=48; salmon vs no-fish n=41)

Table 5.8 Concentration and composition of IDL and LDL sub-fractions

		Baseline	Salmon	Mean difference (95 CI)		No-fish	Mean Difference (95% CI)	p
		Mean ± SD	Mean ± SD	baseline - salmon	p	Mean ± SD	Salmon - no-fish	
IDL:	Mass (mg/dl)	37.82 ± 13.99	31.23 ± 14.61	6.59 (1.69, 11.49)	0.009	34.25 ± 17.76	-1.91 (-8.46, 4.64)	0.559
	Composition:							
	Protein (%)	36.04 ± 3.23	34.03 ± 5.01	2.01 (0.36, 3.66)	0.018	34.74 ± 4.41	-0.44 (-2.27, 1.38)	0.626
	Free Cholesterol (%)	9.48 ± 0.73	9.81 ± 1.03	-0.33 (-0.67, 0.02)	0.061	9.58 ± 0.81	0.15 (-0.22, 0.52)	0.420
	Cholesteryl ester (%)	22.37 ± 1.70	23.14 ± 3.92	-0.77 (-1.99, 0.46)	0.213	22.57 ± 1.62	0.74 (-0.61, 2.10)	0.275
	Triglyceride (%)	19.99 ± 2.01	19.14 ± 2.45	0.86 (-0.06, 1.77)	0.067	19.34 ± 2.11	-0.19 (-1.13, 0.74)	0.677
	Phospholipid (%)	12.12 ± 3.87	13.89 ± 4.43	-1.77 (-3.24, -0.30)	0.019	13.78 ± 4.74	-0.25 (-2.09, 1.59)	0.783
LDL:	Mass (mg/dl)	191.94 ± 52.76	196.16 ± 51.15	-4.21 (-14.46, 6.03)	0.412	194.89 ± 54.65	-2.09 (-16.95, 12.77)	0.778
	Composition:							
	Protein (%)	40.65 ± 1.78	39.83 ± 1.81	0.82 (0.20, 1.43)	0.010	39.81 ± 1.96	0.04 (-0.66, 0.74)	0.911
	Free Cholesterol (%)	8.97 ± 0.57	9.20 ± 0.63	-0.24 (-0.44, -0.04)	0.022	9.00 ± 0.66	0.17 (-0.06, 0.41)	0.147
	Cholesteryl ester (%)	21.39 ± 0.75	21.57 ± 0.93	-0.18 (-0.48, 0.13)	0.247	21.52 ± 0.80	0.14 (-0.22, 0.51)	0.433
	Triglyceride (%)	24.21 ± 1.54	24.34 ± 1.32	-0.13 (-0.73, 0.48)	0.673	24.47 ± 1.68	-0.27 (-0.85, 0.32)	0.362
	Phospholipid (%)	4.79 ± 1.07	5.06 ± 1.21	-0.28 (-0.50, -0.05)	0.017	5.20 ± 1.25	-0.08 (-0.47, 0.30)	0.661

Values are provided as mean ± SD as well as the paired differences (mean difference with 95% CI)
P-values are for paired t-tests (baseline vs salmon n=48; salmon vs no-fish n=41)

Table 5.9 Concentration and composition of VLDL sub-fractions

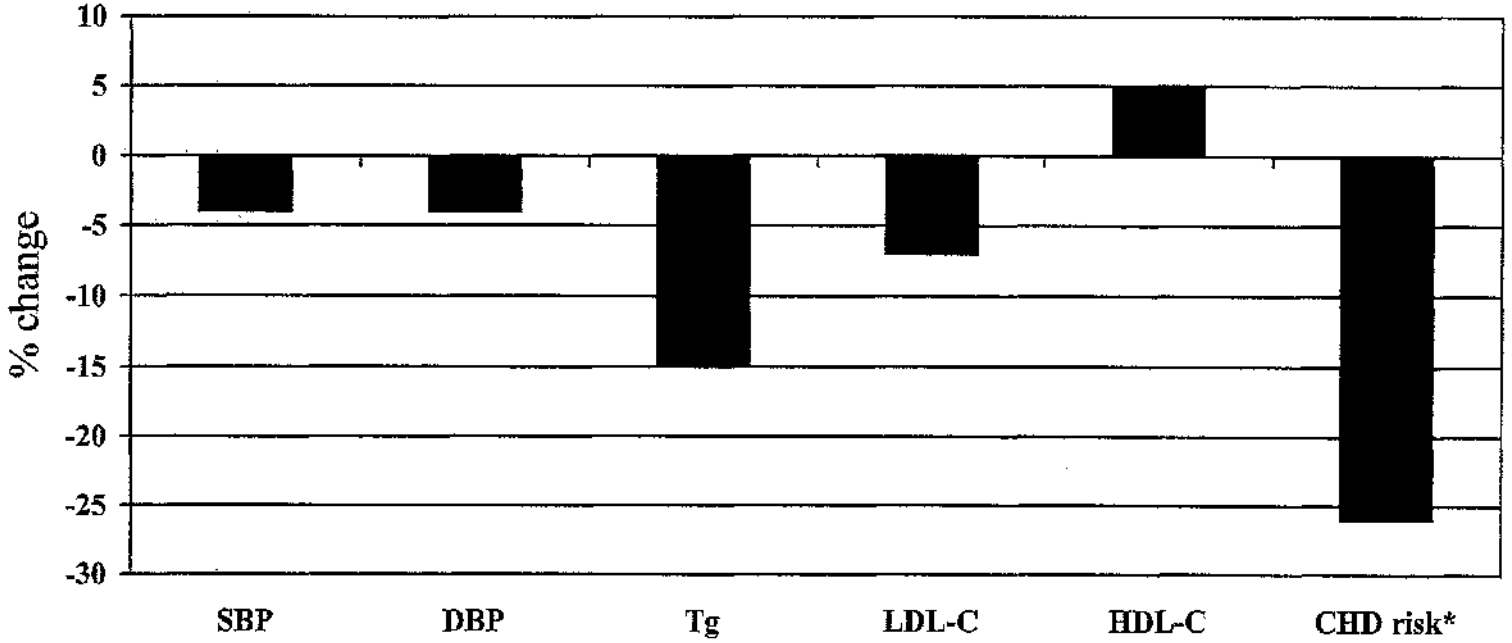
	Baseline Mean ± SD	Salmon Mean ± SD	Mean difference (95 CI) baseline - salmon	p	No-fish Mean ± SD	Mean Difference (95% CI) Salmon - no-fish	p
VLDL-1:Mass (mg/dl)	35.23 ± 25.27	24.51 ± 21.48	10.71 (5.28, 16.15)	0.000	33.30 ± 19.89	-6.81 (-14.30, 0.69)	0.074
Composition:							
Protein (%)	7.46 ± 2.67	6.95 ± 5.55	0.51 (-1.12, 2.13)	0.534	7.44 ± 3.44	0.00 (-1.63, 1.63)	1.000
Free Cholesterol (%)	6.39 ± 0.98	6.31 ± 2.07	0.09 (-0.61, 0.78)	0.804	6.29 ± 1.20	0.26 (-0.50, 1.01)	0.494
Cholesteryl ester (%)	16.38 ± 1.39	16.35 ± 6.78	0.03 (-1.97, 2.03)	0.978	16.12 ± 2.17	-0.37 (-2.02, 1.29)	0.658
Triglyceride (%)	8.31 ± 2.67	7.50 ± 2.76	0.81 (-0.27, 1.88)	0.137	8.15 ± 2.53	-0.47 (-1.62, 0.67)	0.407
Phospholipid (%)	61.46 ± 3.53	62.89 ± 9.43	-1.43 (-4.10, 1.24)	0.287	62.00 ± 4.72	0.58 (-2.36, 3.52)	0.692
VLDL-2:Mass (mg/dl)	24.91 ± 12.14	18.92 ± 11.96	5.99 (2.65, 9.33)	0.001	26.07 ± 12.34	-5.47 (-9.42, -1.51)	0.008
Composition:							
Protein (%)	19.00 ± 3.73	16.20 ± 4.56	2.80 (1.50, 4.10)	0.000	18.57 ± 4.53	-2.12 (-3.53, -0.70)	0.004
Free Cholesterol (%)	8.19 ± 0.71	8.50 ± 1.34	-0.31 (-0.73, 0.11)	0.142	8.16 ± 0.75	0.27 (-0.15, 0.70)	0.200
Cholesteryl ester (%)	20.51 ± 1.31	20.73 ± 2.57	-0.22 (-0.96, 0.51)	0.544	20.94 ± 2.03	-0.08 (-1.09, 0.94)	0.876
Triglyceride (%)	14.60 ± 2.10	13.68 ± 2.78	0.91 (-0.26, 2.09)	0.125	14.01 ± 1.67	-0.32 (-1.28, 0.64)	0.503
Phospholipid (%)	37.71 ± 4.98	40.89 ± 6.81	-3.18 (-5.44, -0.93)	0.007	38.31 ± 5.15	2.24 (0.01, 4.48)	0.049

Values are provided as mean ± SD as well as the paired differences (mean difference with 95% CI)
P-values are for paired t-tests (baseline vs salmon n=48; salmon vs no-fish n=41)

Table 5.10 Effect of compliance to eat fish on blood pressure and plasma lipids

	Compliers (n=36)				Non-compliers (n=6)			
Characteristic	**Salmon**	**No-fish**	**Mean difference (95 CI)**	**p**	**Salmon**	**No-fish**	**Mean difference (95 CI)**	**P**
Blood Pressure (mmHg)								
Sitting systolic	110.5 ± 9	115.3 ± 8	-4.8 (-7, -2)	0.001	108.0 ± 11	110.0 ± 12	-2 (-4, -4)	0.593
Sitting diastolic	71.5 ± 6	74.8 ± 8	-3.3 (-5.5, 1.2)	0.004	72.0 ± 10	72.0 ± 11	0.1 (-5.2, 5.0)	0.977
Mean arterial BP sitting	84 ± 6	88 ± 7	-4 (-6, -2)	0.000	84 ± 9	85 ± 8	-1.0 (-6, 6)	0.793
Triacyglycerol (mmol/l)	0.80 ± 0.38	0.92 ± 0.39	-0.12 (-0.24, 0.01)	0.020	0.61 ± 0.09	0.84 ± 0.13	-0.12 (-0.24, 0.01)	0.042
VLDL (mmol/l)	0.40 ± 0.17	0.45 ± 0.18	-0.05 (-0.11, 0.01)	0.019	0.28 ± 0.14	0.38 ± 0.06	-0.1 (-0.16, -0.04)	0.042
LDL (mmol/l)	2.53 ± 0.69	2.66 ± 0.58	-0.63 (-0.27, 0.01)	0.029	1.76 ± 0.81	1.85 ± 0.52	-0.08 (-0.53, 0.36)	0.616
HDL (mmol/l)	1.44 ± 0.30	1.35 ± 0.28	0.09 (0.01, 0.17)	0.019	1.49 ± 0.35	1.49 ± 0.34	0.00 (-0.26, 0.28)	0.939
Adiponectin ((ug/ml)/1000)	7.98 ± 0.00	7.29 ± 0.00	0.69 (0., 0.)	0.010	7.55 ± 1.59	6.80 ± 1.51	0.75 (0.00, 0.00)	.420
Plasma EPA(μmol/l)	138.1 ± 53.7	29.8 ± 18.6	108 (91, 126)	0.000	52.0 ± 66.7	58.0 ± 68.9	-6 (-32, 20)	.555
Plasma DHA(μmol/l)	160.0 ± 51.1	73.6 ± 32.1	87 (73, 101)	0.000	112.0 ± 47.0	105.0 ± 49.0	7 (42.6, 57.0)	.684

Figure 1. Percentage of change in traditional risk factors and PROCAM-derived CHD risk (salmon vs no-fish; n=41)



SBP, systolic blood pressure; DBP, Diastolic blood pressure; Tg, triglycerides; LDL-C, Low Density Lipoprotein cholesterol, HDL-C, High Density Lipoprotein Cholesterol

* PROCAM-derived CHD risk reduction

Chapter 6

Dietary mis-reporting in weight-stable subjects during an experimental trial, and influences on biomedical outcomes

This chapter has not yet been published. It is presented in the format of a paper intended to be submitted to the British Journal of Nutrition

6.0 Context of the present chapter

This study was designed to answer in part the research questions in section 5.5.

Research questions

1. What is the prevalence of mis-reporting in these subjects?

2. Does misreporting relate to compliance with the intervention?

3. If mis-reporters or non-compliers are excluded from the analysis, how are the conclusions affected?

ABSTRACT

BACKGROUND The prevalence of the inclination towards dietary mis-reporting and also actual mis-reporting (i.e. EI:BMR <1.2 or 1.35) was the main focus of this study.

METHODS 48 non-obese healthy men and women aged 20-55 yrs participated in a study testing the effects of consuming 125 grams/day of salmon for 4 weeks followed by 4 weeks with no-fish on markers of metabolic syndrome. Blood pressure, body composition, and eating behaviour were obtained before and after each intervention period. Food intake was recorded during 7 non-consecutive days including weekends using household measures. Subjects were instructed to maintain their usual eating habits and physical activity patterns during the period of study to avoid weight changes.

RESULTS Three different manoeuvres were explored as possible ways to identify and exclude bias associated with veracity. On average, plasma EPA rose by 96 μmol/ml but 6 subjects showed changes under 20 μmol/ml. 11 subjects had reported EI <1.2 x BMR and 16 subjects reported an intentional inclination to misreport. Dietary mis-reporters complied with fish consumption and therefore were benefited in their metabolic profile in the same way as acceptable reporters. Statistical significance was not affected because of the smaller sample size of the group.

CONCLUSIONS Declared inclination to mis-report is common among non-obese healthy subjects, but intentional misreport does not accurately reflect the actual reporting of implausible diets.

KEY WORDS Diet, Oil-rich fish, Cardiovascular risk factors

6.1 INTRODUCTION

Dietary mis-reporting is a pervasive problem affecting the validation of dietary studies as well as confusing the relationships diet and health (Rosell *et al.*, 2003). By definition it can be described as reporting improbably low (under-reporting) or high (over-reporting) food intakes compared with a standard of reference, usually a physiological measure e.g. doubly labelled water method, or the energy intake to basal metabolic rate ratio (EI:BMR) in the case of energy intake. Urinary excretion of nitrogen provides a standard of reference for dietary protein. Most dietary surveys have found under-reporting to be more frequent than over-reporting (Johansson *et al.*, 1998). Dietary under-reporting can be characterised by under-recording i.e. incomplete recording of foods consumed during the period of study without body weight changes; or by under-eating i.e. decreased food consumption during the recording period with consequent losses in body weight.

Recent studies show that under-eating accounted for all dietary mis-reporting in a group of normal weight female dietitians (Goris and Westerterp, 1999) and accounted for two thirds of all misreporting by obese men (Goris *et al.*, 2000). Subjects mis-reporting food consumption have been shown to do so by different dietary methods (Black *et al.*, 1995) and consistently across time (Black and Cole, 2001; Bandini *et al.*, 2003).

There are suggestions of systematic dietary mis-reporting which suggest intentional under-reporting e.g. for fat (Goris *et al.*, 2000) but the possibility of intentional mis-reporting has not been studied prior to this thesis.

Inclination towards dietary mis-reporting and also actual mis-reporting (i.e. EI:BMR <1.2 or 1.35) was assessed in a group of healthy men and women participating in a trial assessing the effects of eating oil-rich fish.

6.2 METHODS

The methodology described in this chapter was the same as described previously in chapter 5.

6.2.1 Dietary mis-reporting

Dietary under-reporters were identified by means of the energy intake to basal metabolic rate ratio (EI:BMR) (Goldberg *et al.*, 1991). Basal metabolic rate was estimated with the equations recommended by the UK Department of Health (1991). The cut-off points chosen to estimate the prevalence of mis-reporters were EI:BMR <1.2 and <1.35 since these have been the most commonly reported in the literature (Macdiarmid and Blundell, 1998).

Decreased food consumption or under-eating was identified by the presence of significant weight loss during the intervention and control periods (28-days each). Failing to record food intake or under-recording was assumed in those subjects under-reporting food intake without significant weight changes during the 28-d period.

Inclination to misreport food intake was assessed through a questionnaire previously used (Lara *et al.*, 2004).

6.2.3 Statistical analysis

Analysis of variance for repeated measures was performed to compare changes over time taking a significance level of 0.05. Chi-square was used for assessing relationship between categorical variables. In addition the Bland and Altman method was used to compare the presence of mis-reporters, as determined by the EI:BMR, during fish and

no-fish periods. All statistical comparisons were carried out with SPSS 9.0 for windows (SPSS Inc., Chicago, Ill, USA).

6.3 Results

The characteristics of the group under study are shown in **table 6.1**. On average the group was young; only two participants were above 40yrs of age. Normal weight subjects predominated with only one-fourth of the sample classified as overweight; obese subjects were not studied. Average BMI was 23.2 kg/m^2. The proportion of current drinkers was high, while a substantial proportion of the participants were never-smokers. All these factors reflect relatively high educational levels. Self-reported physical activity patterns showed a homogeneous high activity level in different areas (housework, work, and sports and exercise). Eating behaviour was within normal ranges as indicated by dietary restraint, disinhibition and hunger scores of the three-factor eating questionnaire. The self-reported nutrient intake during the fish consumption intervention period is shown in **table 6.2**. During the fish consumption intervention period self-reported food intake represented 7938.7 ± 2298.0 kilojoules/day comprising 46.2 ± 8.2% of energy as carbohydrates, 17.9 ± 5.3 % as protein, 34.5 ± 7 % as fat and 4.3 ± 4.5% as alcohol. Mean daily consumptions of sodium and potassium were 99.2 ± 32.7 and 75.4 ± 22.4 mmols, respectively.

The maintenance of a neutral energy balance as indicated by constant average body weight and body composition, as assessed by body fat or waist circumference, during the intervention and control periods exclude the possibility of under-eating as a group (**Table 6.3**). The overall EI:BMR ratio for the fish and no-fish consumption periods as well as the prevalence of under-reporting by these subjects are shown in **table 6.4**. No significant differences between study period, either in the mean values for EI:BMR or

in the prevalence of mis-reporters by any of the cut-off point, was observed. **Figure 6.1** shows the correspondence in EI:BMR ratios during the different phases of the study; a bland and Altman analysis is shown.

On average subjects inclined to mis-report presented lower EI:BMR than those inclined to report accurately (addressed by means of a questionnaire), however such differences were not statistically significant (**Table 6.4**). Subjects inclined to mis-report by questionnaire and those identified as mis-reporters through the EI:BMR ratio were not exactly the same, however a considerable proportion of those who say they will under-report, quite often do so (50% actually reported EI:BMR <1.35). But this match was not perfect and indeed 40 % of those who said they were inclined to over-report actually under-reported based on EI:BMR<1.35. The small number of subjects identified as non-compliant with fish consumption was not consistently associated with intentional mis-reporting (**Table 6.5 to 6.7**), however 3 out of 5 non-compliant subjects were inclined to mis-report.

The differences in EPA (**Table 6.8**) triglycerides (**Table 6.9**), blood pressure (**Table 6.10**) and adiponectin (**Table 6.11**) showed the similar patterns of change among mis-reporters; however most of these differences were not statistically significant given the smaller numbers in the subgroups formed according to mis-reporting.

6.4 Discussion

Neglected in the past, dietary mis-reporting has recently attracted the attention of researchers from different fields. With a significant extent and an unclear impact on the diet and health/disease relationships, dietary misreporting is a rather difficult problem to solve. Several studies have shown that dietary mis-reporting is an insidious problem to most dietary surveys independently of the dietary methodology employed (Black and

Cole, 2001) and it is influenced by several social factors, eating behaviour, as well as demographic characteristics (Macdiarmid and Blundell, 1998). In addition, several studies discriminating between the presence of under-eating or under-recording demonstrate the complexity behind such a problem and the discouraging scenery faced by most nutritionists dealing with this problem.

6.4.1 Food consumption under-recording more ubiquitous prevalent than under-eating

In the present study dietary mis-reporting was assessed in participants of an experimental study consuming salmon daily during 28 days and no-fish for another 28-day period. These subjects showed a stable body weight (65.5, 65.6 and 65.7 kg at baseline, salmon and no-fish periods respectively) during the length of the study indicating absence of under-eating, in contrast to findings from other studies (Goris and Westerterp, 1999). Therefore, we assume that under-recording of the food consumed, whether by missing the recording of some foods or recording low amount of food eaten, explained dietary under-reporting in its totality.

6.4.2 Dietary mis-reporting was consistent over time

In the present study the magnitude of mis-reporting as indicated by the EI:BMR ratio and the prevalence of mis-reporters by two different cut-off points (<1.2 and <1.35) was consistent between the two phases of the study (e.g. salmon 23% vs No-fish 21% for an EI:BMR<1.2). A closer observation of the results showed that individual subjects who mis-reported were persistent over time (**Figure 6.1**). Ten out of 11 subjects reporting a EI:BMR ratio less than 1.2 on the first time, did so again on the second time; at a EI:BMR <1.35, 19 out of 20 subjects mis-reported in both occasions. These results

are in agreement with the findings from previous studies showing that mis-reporters did so in four different times during a year (Black and Cole 2001).

6.4.3 Effects of mis-reporting on clinical parameters

This study compared the effects of mis-reporting in clinical and metabolic variables. A small number of people (n=6) was identified as non-compliant with fish-consumption. However, these did not show any particular characteristic compared to the rest of the group. Therefore comparisons were confined to inclination to mis-report and subjects with low EI:BMR. Results showed that even though these subjects are openly inclined to mis-report their food intake and or to report low EI:BMR, they still might be able to comply with other requests from the study (e.g. eating fish, maintaining body weight, eating patterns, attending visits) and show the effects expected (decrease in blood pressure, increase in EPA) (see **Tables 6.6.-6.9**).

6.4.4 Who are those inclined to mis-report?

Inclination to mis-report assessed by single and simple questions has been reported previously in a group of women and related to body mass index and eating behaviour (Lara *et al.*, 2004 Chapter 2). In the present study inclination to mis-report was prominent (58% declared an intention to either under- or over-report their food intake). On average subjects inclined to under-report tended to be younger (27y) than over- (33y) and accurate reporters (28y) (P=0.051). Accurate reporters presented a BMI of 22.8 compared to under- (23.4) and over-reporters (23.5); however there were no significant differences (P=0.757).

Subjects inclined to under-report indeed had the lowest mean-EI:BMR ratio (1.33) among the three groups (over-reporters 1.37; accurate reporters 1.47), however numbers

in these subgroups were small and differences were not significant. These results suggest that declaring an inclination to under-report might indeed identify subjects reporting low EI:BMR ratios but the false positive rate is high.

6.4.5 Who are the under-reporters (EI:BMR <1.2)?

On average, under-reporters had a significantly higher BMI than acceptable reporters (24.9 ± 3.8 vs 22.7 ± 2.7; P=0.034). No significant difference in age (under-reporters 30y and acceptable reporters 28y) was observed. On average 37.5% of subjects with an EI:BMR<1.2 declared an inclination to report less (under-report); that figure raised to 50% among subjects with an EI:BMR<1.35.

6.4.6 Who are the non-compliers?

The small number of non-compliers (n=5) with fish consumption were not obviously different from the rest of the participants. On average their age was 27 years, BMI 23.3 ± 2.8, and mean EI:BMR 1.42 ± 0.24. Three of them reported EIBMR >1.35, one <1.35 and one < 1.2. Two of the non-compliers reported an inclination to report accurately, presenting EI:BMR >1.5. One non-complier reported an inclination to under-report, presenting a EI:BMR <1.2. The two remaining subjects reported an inclination to over-report; one of them had a EI:BMR <1.35 (but >1.2) and the other one >1.35.

Subjects whose EPA failed to rise, also failed to show any effect on blood pressure or triglycerides (**Tables 6.7, 6.8**) which strengthens the view that they did not eat the fish.

Several studies have shown a relationship between social desirability and dietary under-reporting (Taren *et al.*, 1999; Hebert *et al.*, 1997). Given that the effect that social desirability represents the intention to comply with norms, it is likely that subjects scoring high on the social desirability scale might not be willing to openly declare an

inclination to mis-report, but still do so in the reporting of food consumption. So, together, results from the present study and evidence from several other studies points out towards the possibility that intentional reasons to mis-report food consumption are, as it was hypothesised, indeed more important that non-intentional reasons.

6.4.7 Conclusion

The results from this work show again that declared inclination to mis-report is common among non-obese healthy subjects willing to undertake an intervention diet study, but extends that knowledge with the finding that intentional misreport does not accurately reflect the actual reporting of implausible diets.

Lack of veracity in dietary reporting does not identify individuals who failed to comply with instructions to eat fish. Finally excluding mis-reporters does not change the pattern of results, so no bias was introduced.

If the subjects declaring an intention to misreport were to be excluded at recruitment, large numbers of subjects would have to be excluded, possibly introducing bias through loss of representativeness. There would then be fewer under-reporters and dietary compliance would be a little better. Larger studies are needed to establish whether this would be a useful manoeuvre for dietary surveys in general.

Table 6.1 Baseline characteristics in 48 subjects

Characteristic	Mean ± SD
Age (yrs)	28.2 ± 7.8
Height (m)	1.68 ± 0.09
Weight (kg)	65.4 ± 10.4
Waist (cm)	75.6 ± 7.4
Hip (cm)	96.6 ± 7.6
BMI (kg/m2)	23.2 ± 3.1
Overweight (BMI 25-29.9) (%)	25
Women (%)	66.6
Smoking habits (%)	
Current smoker	20
Former smoker	8
Never smoker	66
Current drinkers (%)	84
Eating behaviour scores	
Dietary restraint	7.2 ± 4.2
Disinhibition	5.3 ± 3.1
Perceived hunger	4.6 ± 2.3

Values are mean and SD unless otherwise stated

Table 6.2 Self-reported daily nutrient intakes in 48 subjects during the salmon period

Characteristic	Mean ± SD
Energy (Kilojoules)	7938.7 ± 2298.0
(Kilocalories)	1888.7 ± 546.3
Carbohydrates (%)	46.2 ± 8.2
Protein (%)	17.9 ± 5.3
Fat (%)	34.5 ± 7
Poly-unsaturated fatty acids (%)	6.1 ± 1.9
Mono-unsaturated fatty acids (%)	12.2 ± 3.3
Saturated fatty acids (%)	11.1 ± 2.7
Alcohol (%)	4.3 ± 4.5
Sodium mmols	99.2 ± 32.7
(mg)	(2281.3 ± 751.5)
Potasium mmols	75.4 ± 22.4
(mg)	(2939.3 ± 872.8)
Cholesterol	252.3 ± 139.4

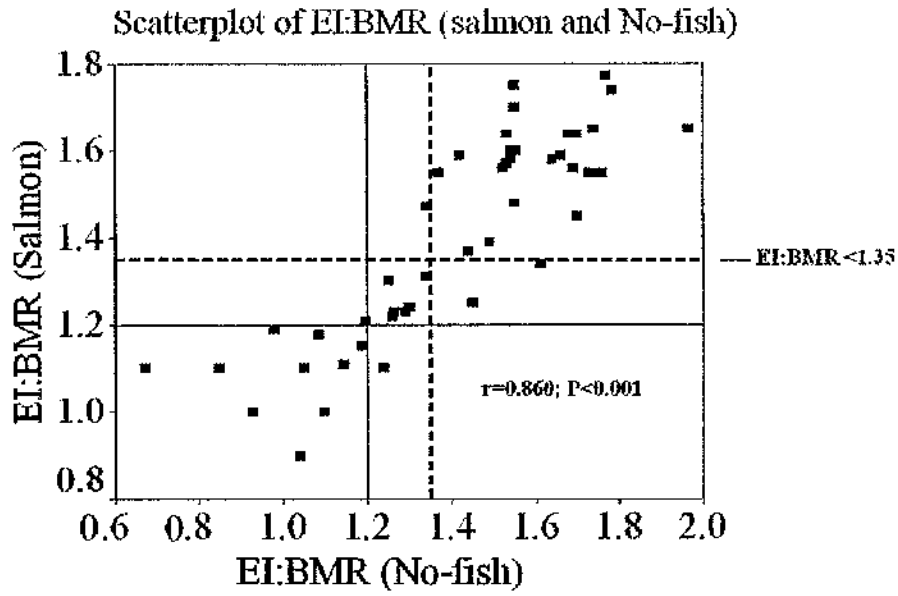
Table 6.3 Body weight and composition changes

Characteristic	Baseline n=48	Post intervention n=48	1 month later n=41
Body weight (kg)	65.5 ± 10.4	65.6 ± 10.4	65.7 ± 10.5
Waist (cm)	75.6 ± 7.4	75.5 ± 7.7	75.6 ± 7.5
Body fat (%)	20.1 ± 8.5	20.0 ± 8.5	20.1 ± 8.9

Table 6.4 EI:BMR ratio and prevalence of under-reporters

Characteristic	Salmon consumption period n=48	No-fish consumption period n=48	P
EI:BMR	1.40 (1.32, 1.48)	1.42 (1.33, 1.50)	0.448
EI:BMR <1.2 n (%)	11 (22.9)	10 (20.8)	
EI:BMR ≥1.2 n (%)	37 (77.1)	38 (79.2)	0.788
EI:BMR < 1.35 n (%)	20 (41.7)	19 (39.6)	
EI:BMR ≥ 1.35 n (%)	28 (58.3)	29 (60.4)	0.675

EI:BMR values represent mean (95% CI). P-value corresponds to student t-test analysis



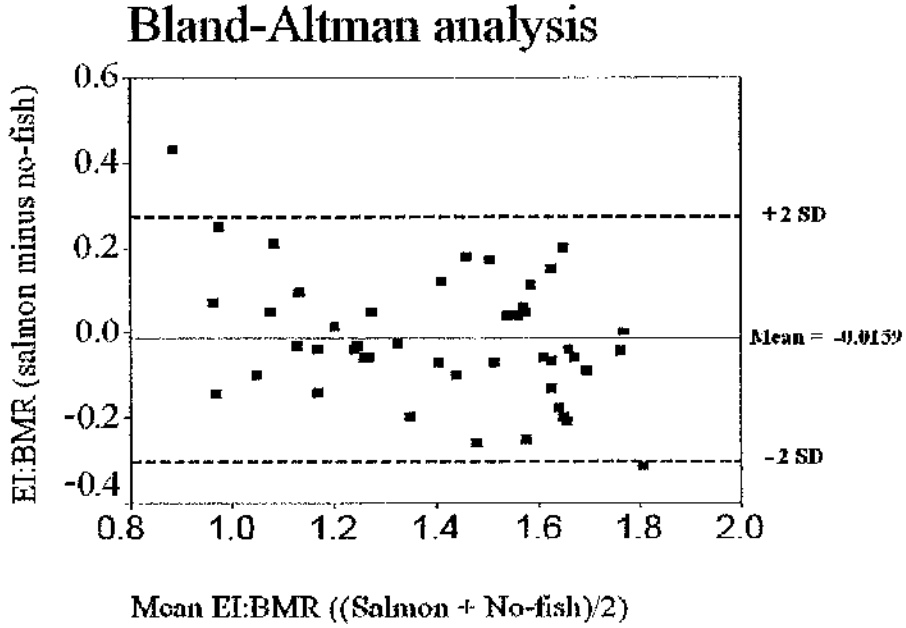


Figure 6.1 Subjects who under-reported once (on salmon) were very likely to do so a second time (no-salmon). There was no bias according to EI:BMR

Table 6.5 Who are the intentional mis-reporters?

Inclination to:	**Over-report n=12**	**Under-report n=16**	**Accurate-report n=20**	**P**
EI:BMR (mean±SD) (n=48)	1.37 ± 0.31	1.33 ± 0.24	1.47 ± 0.27	[a]0.322
	n (%)	n (%)	n (%)	
EI:BMR <1.2 (n=11)	2 (18)	6 (55)	3 (27)	
EI:BMR >1.2 (n=37)	10 (27)	10 (27)	17 (46)	[b]0.234
EI:BMR <1.35 (n=20)	5 (25)	8 (40)	7 (35)	
EI:BMR >1.35 (n=28)	7 (25)	8 (29)	13 (46)	[b]0.663
Fish compliant (n=43)	10 (23)	15 (35)	18 (42)	
Fish non-compliant (n=5)	2 (40)	1 (40)	2 (40)	[b]0.788

[a] ANOVA Analysis; [b] Chi-square

Table 6.6 Who are the under-reporters?

	EI:BMR		P	EI:BMR		P
	<1.2 (n=11)	**>1.2 (n=37)**		**<1.35 (n=20)**	**>1.35 (n=28)**	
Inclination to:	n (%)	n (%)		n (%)	N (%)	
Over-report n=12	2 (17)	10 (83)		5 (42)	7 (58)	
Under-report n=16	6 (38)	10 (62)		8 (50)	8 (50)	
Accurate-report n=20	3 (15)	17 (85)	0.234	7 (35)	13 (65)	0.663
Fish-compliant (n=43)	10 (23)	33 (77)		18 (42)	25 (58)	
Fish non-compliant (n=5)	1 (20)	4 (80)	0.679	2 (40)	3 (60)	0.660

P-value for Chi-square test

Table 6.7 Who are the non-compliers (no rise in EPA)?

	Fish-compliant (EPA rise >20) (n=43)	**Fish non-compliant (EPA rise<20) (n=5)**	**P**
Inclination to:			
Over-report (n=12)	10 (83)	2 (17)	
Under-report (n=16)	15 (94)	1 (6)	
Accurate-report (n=20)	18 (90)	2 (10)	0.788
EI:BMR <1.2 (n=11)	10 (91)	1 (9)	
EI:BMR >1.2 (n=37)	33 (89)	4 (11)	0.234
EI:BMR <1.35 (n=20)	18 (90)	2 (10)	
EI:BMR >1.35 (n=28)	25 (89)	3 (11)	0.663

P-value for Chi-square test

Table 6.8 Effect of mis-reporting on plasma EPA concentrations (μmol/L)

Inclination to:	**EPA Baseline**	**EPA Salmon**	**EPA No-fish**	**P**
Over-report (n=11)	46.2 ± 21.8	132.2 ± 71.3	40.9 ± 22.3	0.001
Under-report (n=13)	26.1 ± 14.5	124.8 ± 54.1	25.4 ± 11.3	0.001
Report accurately (n= 16)	21.9 ± 13.7	127.4 ± 63.2	34.2 ± 41.3	0.001
EI:BMR <1.2 (%) (n=10)	38.8 ± 18.2	115.2 ± 59.7	32.2 ± 17.4	0.001
EI:BMR >1.2 (%) (n=30)	27.2 ± 19.0	132.4 ± 62.2	33.5 ± 32.35	0.001
EI:BMR <1.35 (%) (n=16)	32.5 ± 16.9	117.1 ± 61.4	28.3 ± 15.8	0.001
EI:BMR >1.35 (%) (n=24)	28.2 ± 21.3	137.7 ± 61.0	37.6 ± 37.0	0.001
Fish compliant (n=43)	30.2 ± 16.4	131.3 ± 55.6	31.5 ± 27.9	<0.009
Fish non-compliant (n=5)	34.2 ± 22.8	52.0 ± 66.7	58.0 ± 69.0	0.779

P-value for ANOVA analysis

Table 6.9. Effects of mis-reporting and fish compliance on triglycerides (Tg; mmol/L)

	Tg baseline	**Tg salmon**	**Tg no-fish**	**Difference No fish - Salmon**	**P**
Inclination to: Over-report n=10	1.09 ± 0.45	0.86 ± 0.52	0.99 ± 0.45	0.13	0.806
Under-report n=14	0.85 ± 0.25	0.76 ± 0.29	0.91 ± 0.32	0.15	0.306
Report accurately n=20	0.83 ± 0.23	0.69 ± 0.23	0.85 ± 0.34	0.16	0.028
EI:BMR <1.2 (%) (n=10)	0.87 ± 0.30	0.82 ± 0.42	0.90 ± 0.34	0.08	0.99
EI:BMR >1.2 (%) (n=30)	0.90 ± 0.34	0.74 ± 0.33	0.91 ± 0.38	0.17	.016
EI:BMR <1.35 (%) (n=16)	0.79 ± 0.30	0.72 ± 0.35	0.87 ± 0.28	0.15	.121
EI:BMR >1.35 (%) (n=24)	0.98 ± 0.33	0.78 ± 0.35	0.93 ± 0.41	0.15	.121

P-value for student t-test

Table 6.10 Effect of mis-reporting and fish compliance on Systolic blood pressure (mmHg)

	Systolic BP Baseline	Systolic BP Salmon	Systolic BP No-fish	Difference No fish - Salmon	P
Inclination to: Over-report n=10	110 ± 9	109 ± 8	113 ± 10	3.3	0.179
Under-report n=13	117 ± 8	111 ± 9	116 ± 8	4.6	0.011
Report accurately n=14	117 ± 8	111 ± 11	116 ± 8	5.1	0.020
EI:BMR <1.2 (%) (n=10)	118 ± 8	112 ± 11	117 ± 10	5.0	0.040
EI:BMR >1.2 (%) (n=27)	114 ± 9	110 ± 9	115 ± 8	5.0	0.001
EI:BMR <1.35 (%) (n=18)	117 ± 9	111 ± 11	116 ± 10	5.0	0.020
EI:BMR >1.35 (%) (n=19)	113 ± 9	110 ± 7	114 ± 7	4.0	0.010
Fish non-compliant (n=5)	108 ± 15	108 ± 12	111 ± 11	3.0	0.778
Fish compliant (n=37)	115 ± 9	111 ± 9	115 ± 8	4.0	0.0004

P-value for student t-test

Table 6.11 Effect of mis-reporting and fish compliance on plasma adiponectin concentrations (µmol/L)

	Baseline	Salmon	No-fish	Difference No fish - Salmon	P
Inclination to: Over-report n=10	7.85 ± 4.1	9.20 ± 4.19	7.87 ± 3.14	0.59	0.786
Under-report n=13	7.86 ± 3.83	10.40 ± 5.52	8.98 ± 3.19	1.42	0.546
Report accurately n=14	7. 24 ± 3.45	9.97 ± 5.41	8.09 ± 3.31	1.88	0.258
EI:BMR <1.2 (%) (n=10)	9.26 ± 4.61	9.87 ± 4.25	10.20 ± 4.76	0.23	0.864
EI:BMR >1.2 (%) (n=27)	7.02 ± 3.57	8.77 ± 4.31	7.70 ± 3.92	1.07	0.245
EI:BMR <1.35 (%) (n=18)	8.26 ±4.22	8.86 ± 3.67	8.66 ± 4.09	0.20	0.765
EI:BMR >1.35 (%) (n=19)	7.15 ± 3.73	9.27 ± 4.75	8.21 ± 4.30	1.06	0.245
Fish non-compliant (n=5)	6.91± 2.36	7.05 ± 1.59	6.89 ± 1.52	0.58	0.856
Fish compliant (n=37)	7.01 ± 3.87	8.12 ± 4.25	7.22 ± 4.02	0.90	0.098

P-value for student t-test

Chapter 7

General discussion

7.1 Overview of the scope of this thesis

This thesis has explored scientific issues at the heart of dietetics and applied nutrition, as part of training for a PhD degree. A recurrent theme has been that of veracity and mis-reporting of food consumption by volunteer subjects. The results of this research provide new angles from which to consider the association between food exposure and bio-medical outcomes, upon which much of nutritional science depends, and the translation from experimental research into guidelines and dietary advice.

The work followed a number of logical steps presented on separate chapters, in which research questions were raised, and then answered. The process behind this research dissertation was thus an iterative one. The methods employed progressed from a **Systematic Review Process**, to perform a new review of the literature on dietary mis-reporting, **Cross-sectional Survey** methods, for the first time actually asking subjects whether they are inclined to mis-report a classical **Dietary Intake Study** and finally a relatively large-scale **Experimental Crossover-Design Intervention**.

The research questions and their answers are summarised in **table 7.1**.

One simple conclusion from this work is that declared intention to mis-report is common. Its contribution to **under-reporting**, as evidenced by low EI:BMR ratios is small, but people who state an inclination either to under- or over-report both seem to record diets with low EI:BMR ratios. The data available within this thesis are not enough to be entirely

confident but it is easy to ask before starting a dietary intake study and a stated inclination to mis-report may be a reason to review data with scepticism.

Establishing the true intake of foods and nutrients is difficult. For many nutrients there is no objective measures to quantify intake, and for food essentially none. However oil-rich fish consumption is well-known to increase EPA and DHA in tissues such as adipose tissue, blood cell membranes, and circulating concentrations in plasma. Others have used the rise in plasma as a marker of fish-oil consumption but none of the very few published fish eating studies have used any biomarkers. For the present study plasma was used recognising that it reflects rather recent fish consumption better than for example adipose tissue biopsies. It is also much easier and cheaper. The change in concentration varies between individuals, and this probably depends on compliance, at least over the previous few days: the lack of change in serum triglycerides and blood pressure suggests strongly that the non-compliant subjects were non-compliant for much of the study period.

It was not the original intention to focus on fish in this thesis, but the finding that fishmongers showed less under-reporting, ate more fish and had lower blood pressures than butchers or schoolteachers, made it interesting to design an intervention trial using fish. The previous evidence is very limited, although there is consistent evidence that fish-oil supplements improve blood pressure and triglycerides. Recognising the growing importance of metabolic syndrome worldwide (it probably affects 20-30% of all populations, exacerbated by weight gain and specifically a large waist circumference), it was decided to address all the diagnostic features of metabolic syndrome. Most improved with 125 g/day salmon - even in young adults none of whom had metabolic syndrome. The

changes in blood pressure and lipids alone with salmon intake predict around a 25% reduction in CHD risk. It would seem very attractive to examine the effect of fish in treating patients with metabolic syndrome.

Within this experiment it was also interesting to find that serum adiponectin tended (P=0.086) to rise with fish consumption. This strengthens the findings since adiponectin is proposed as part of the mechanism behind metabolic syndrome (Trujillo and Scherer, 2005).

The experimental study was relatively large (n=48) but these numbers were not sufficient to explore fully the relationship between **inclination to mis-report, under-reporting** according to EI:BMR ratio, and **compliance** with instruction to eat salmon every day. The data suggest that all intentional mis-reporters will tend to record low food intakes.

Data on EPA and DHA suggested that 5 of these subjects in this study (10%), despite verbal assurances, did not in fact eat the fish. Since this study was not intended as a test of advice to eat fish (demanding intention-to-treat analysis), it is reasonable to exclude non-compliant subjects to evaluate bio-medical outcomes. Having excluded these presumed non-compliant subjects the analysis on metabolic syndrome components was not strengthened.

Of the 6 subjects judged non-compliant because plasma EPA did not change by over 20 μmol/L (the group mean change being 96), 2 were under-reporters (EI:BMR<1.2); three subjects declared an intention to mis-report.

Thus in conclusion, the evidence was weak but simply asking people whether they would be inclined to mis-report food consumption would allow exclusion of most subjects who proved non-compliant and many who under-reported. Some subjects declaring an intention to mis-report went on to be compliant and to record acceptable energy intakes. When potential subjects are plentiful for dietary studies, it would seem reasonable to exclude those who say they are inclined to mis-report. There does not appear to be any bias introduced into dietary records, but given the high numbers of "mis-reporters" there is a risk that samples will be non-representative. The consistency of under-reporting was assessed from dietary intake analysis during fish and no-fish periods within the study; it appears that under-reporting was rather consistent within individuals.

7.2 LIMITATION OF METHODS USED IN THIS THESIS

1 Further validation of the questions on mis-reporting would be desirable especially test-retest repeatability over large periods.

2 Samples of nurses, obese patients, butchers, fishmongers and teachers were obtained by "convenience" methods and so not necessarily representative of their occupational groups or of their genders.

3 Numbers, especially in subgroups analysis, were often too low for confidence; Beta-type errors are a most frequent problem (i.e. failing to detect a true association) than alpha (i.e. a spuriously high p-value).

4 Because of sensitivity amongst participants about veracity of reporting were not used with butchers, fishmongers or schoolteachers.

5 In the experimental study treatment order was not varied. This decision was based on uncertainty about willingness of subjects to continue for 8 weeks or to complete a 4-week no-fish period, in which case at least a comparison with baseline would have been possible. In the event, drop outs were low, so randomised order would probably have been possible. It is not possible to gauge whether using a fixed order introduced any bias. It might be expected that dietary compliance would decline, which would tend to weaken this study and could conceal real effects.

6 Eating 125g of salmon daily is not reasonable when translated into health promotion. Having proved the beneficial effects of fish, further research needs to address more acceptable advice, and probably in older subjects with features of metabolic syndrome.

7.3 TOPICS OF FUTURE RESEARCH RAISED BY THESE CONCLUSIONS

- Assessment of true prevalence of mis-reporting and association with under-reporting in a large, representative population-based survey including men and women with a range of ages, BMI, etc.

- Explore different wording and format of the questions on inclination to mis-report food consumption. The present format was designed to force a choice or direction, with accurate recording only as a category of "other".

- Study fish consumption in patients with metabolic syndrome

- Studies of less frequent fish consumption and of other accessible fish types.

- Long-term study of frequent fish consumption, with detailed body composition analysis to assess body fat compartments

- Formal insulin sensitivity studies with fish consumption, as the metabolic effects seen appear to relate to insulin action

Table 7.1 Research questions addressed in the present thesis

	Questions	Answers
Chapter 2	1. Do (some) people intentionally provide inaccurate or misleading information about food intake?	Yes, and it seems a common issue, both to over- and under-report
	2. Does a declared inclination to over-report or to under-report relate to BMI, gender, age or psychological variables?	Yes. It is related with BMI, dietary disinhibition, current dieting, and dissatisfaction with current body weight
Chapter 3	1. Do men show different patterns of inclination to mis-report than women?	Could not answer this question, as these men were not asked.
	2. Do people with food related-occupations who might influence food choice of others exhibit different patterns of intentional dietary mis-reporting?	Yes. Fishmongers were better reporters than butchers according to EI:BMR
Chapter 4	1. Do butchers, fishmongers and schoolteachers report quantitatively different diet composition?	Yes Fishmongers ate more fish than the other groups. They also reported eating more biscuits and cakes probably because of their working hours. Differences in other foods and nutrients were similar.
	2. Is there any relationship between diet composition and health indicators in these groups?	Yes. Higher consumption of fish amongst fishmongers was significantly related with a lower blood pressure than amongst butchers or schoolteachers
Chapter 5	1. Does higher consumption of fish reduces blood pressure?	Yes (BP 4-5 mmHg lower on average) when eating 125 g of salmon daily
	2. Does higher consumption of fish modify other indices of metabolic syndrome?	Yes. Fish reduces triglycerides, VLDL and LDL-cholesterol, and increases HDL-cholesterol, predicting a 25% reduction in the risk for CHD.
Chapter 6	1. What is the prevalence of mis-reporting in these subjects	Inclination to mis-report 38%, Under-reporting EI:BMR<1.2 Non-compliers (low EPA rise) 10%
	2. Does misreporting relates to compliance with the intervention	Not closely. Three out of 5 non-compliers misreported food consumption.
	3. If mis-reporters or non-compliers are excluded from the analysis, how are the conclusions affected?	With the small numbers in this part of the study, there was no effect on relationships with metabolic syndrome variables

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Appendix 1
The Rosenberg Self-esteem scale (Rosenberg, 1965)

Below is a list of statements dealing with your general feelings about yourself. If you strongly agree, circle SA. If you agree with the statement, circle A. If you disagree, circle D. If you strongly disagree, circle SD.

		1. Strongly Agree	2 Agree	3. Disagree	4. Strongly Disagree
A)	On the whole, I am satisfied with myself.	SA	A	D	SD
B)	At times I think I am no good at all.	SA	A	D	SD
C)	I feel that I have a number of good qualities.	SA	A	D	SD
D)	I am able to do things as well as most other people.	SA	A	D	SD
E)	I feel I do not have much to be proud of.	SA	A	D	SD
F)	I certainly feel useless at times.	SA	A	D	SD
G)	I feel that I'm a person of worth, at least on an equal plane with others.	SA	A	D	SD
H)	I wish I could have more respect for myself.	SA	A	D	SD
I)	All in all, I am inclined to feel that I am a failure.	SA	A	D	SD
J)	I take a positive attitude toward myself.	SA	A	D	SD

Appendix 2

Psychological well being questionnaire (Bradley and Gamsu, 1994)

Please circle a number on each of the following scales to indicate how often you feel each phrase has applied to you in the past few weeks:

		All the time	↔		Not at all
A)	I feel that I am useful and needed	3	2	1	0
B)	I have crying spells or feel like it	3	2	1	0
C)	I find I can think quite clearly	3	2	1	0
D)	My life is pretty full	3	2	1	0
E)	I feel downhearted and blue	3	2	1	0
F)	I enjoy the things I do	3	2	1	0
G)	I feel nervous and anxious	3	2	1	0
H)	I feel afraid for no reason at all	3	2	1	0
I)	I get upset easily or feel panicky	3	2	1	0
J)	I feel like I'm falling apart and going to pieces	3	2	1	0
K)	I feel calm and can sit still easily	3	2	1	0
L)	I fall asleep easily and get a good night's rest	3	2	1	0
M)	I feel energetic, active or vigorous	3	2	1	0
N)	I feel dull or sluggish	3	2	1	0
O)	I feel tired, worn out, used up, or exhausted	3	2	1	0
P)	I have been waking up feeling fresh and rested	3	2	1	0
Q)	I have been happy, satisfied, or pleased with my personal life	3	2	1	0
R)	I have felt well adjusted to my life situation	3	2	1	0
S)	I have lived the kind of life I wanted to	3	2	1	0
T)	I felt eager to tackle my daily tasks or make decisions	3	2	1	0
U)	I have felt I could easily handle or cope with any serious problem or major change in my life	3	2	1	0
V)	My daily life has been full of things that were interesting to me	3	2	1	0

Appendix 3
Three-factor eating questionnaire (Stunkard and Messick, 1985)

Please answer True of False by circling the appropriate characters (T or F).

		True	False
A)	When I smell a sizzling steak or see a juicy piece of meat, I find it very difficult to keep from eating it, even if I have just finished a meal.	T	F
B)	I usually eat too much at social occasions, like parties and picnics.	T	F
C)	I am usually so hungry that I eat more than three times a day	T	F
D)	When I have eaten my quota of calories, I am usually good about not eating any more.	T	F
E)	Dieting is so hard for me because I just get too hungry.	T	F
F)	I deliberately take small helpings as a means of controlling my weight.	T	F
G)	Sometimes things just taste so good that I keep on eating even when I am no longer hungry.	T	F
H)	Since I am often hungry, I sometimes wish that while I am eating, an expert would tell me that I have had enough or that I can have something more to eat.	T	F
I)	When I feel anxious, I find myself eating.	T	F
J)	Life is too short to worry about dieting	T	F
K)	Since my weight goes up and down, I have gone on reducing diets more than once	T	F
L)	I often feel so hungry that I just have to eat something.	T	F
M)	When I am with someone who is overeating, I usually overeat too.	T	F
N)	I have a pretty good idea of the number of calories in common food.	T	F
O)	Sometimes when I start eating, I just can't seem to stop.	T	F
P)	It is not difficult for me to leave something on my plate.	T	F
Q)	At certain times of the day, I get hungry because I gotten used to eating then.	T	F
R)	While on a diet, if I eat food that is not allowed, I consciously eat less for a period of time to make up for it.	T	F
S)	Being with someone who is eating often makes me hungry enough to eat also.	T	F
T)	When I feel blue, I often overeat.	T	F

		True	False
U)	I enjoy eating too much to spoil it by counting calories or counting my weight.	T	F
V)	When I see a real delicacy, I often get so hungry that I have to eat right away.	T	F
W)	I often stop eating when I am not really full as a conscious mean of limiting the amount that I eat.	T	F
X)	I get so hungry that my stomach often seems like a bottomless pit.	T	F
Y)	My weight has hardly changed at all in the last ten years.	T	F
Z)	I am always hungry so it is hard for me to stop eating before I finish the food on my plate.	T	F
AA)	When I feel lonely, I console myself by eating.	T	F
AB)	I consciously hold back at meals in order not to gain weight.	T	F
AC)	I sometimes get very hungry late in the evening or at night.	T	F
AD)	I eat anything I want, anytime I want.	T	F
AE)	Without even thinking about it, I take a long time to eat.	T	F
AF)	I count calories as a conscious means of controlling my weight.	T	F
AG)	I do not eat some foods because they make me fat.	T	F
AH)	I am always hungry enough to eat at any time.	T	F
AI)	I pay a great deal of attention to changes in my figure.	T	F
AJ)	While on a diet, if I eat a food that is not allowed, I often then splurge (binge) and eat other high calorie foods.	T	F

Directions: Please answer the following questions by circling the number that is appropriate to you

A)	How often are you dieting in a conscious effort to control your weight?			
	1 rarely	2 sometimes	3 usually	4 always
B)	Would a weight fluctuation of 5 lbs affect the way you live your life?			
	1 not at all	2 slightly	3 moderately	4 very much
C)	How often do you feel hungry?			
	1 only at mealtimes	2 sometimes between meals	3 often between meals	4 almost always
D)	Do your feelings of guilt about overeating help you to control your food intake?			
	1 never	2 rarely	Often	4 always
E)	How difficult would it be for you to stop eating halfway through dinner and not eat for the next four hours?			
	1 easy	2 slightly difficult	3 moderately difficult	4 very difficult
F)	How conscious are you of what you are eating?			

1 not at all 2 slightly 3 moderately 4 very much

G) How frequently do you avoid 'stocking up' on tempting foods?

1 almost never 2 seldom 3 usually 4 almost always

H) How likely are you to shop for low calorie foods?

1 unlikely 2 slightly unlikely 3 moderately likely 4 very likely

I) Do you eat sensibly in front of others and splurge alone?

1 never 2 rarely 3 often 4 always

J) How likely are you to consciously eat slowly in order to cut down on how much you eat?

1 unlikely 2 slightly unlikely 3 moderately likely 4 very likely

K) How frequently do you skip dessert because you are no longer hungry?

1 almost never 2 seldom 3 at least once a week 4 almost every day

L) How likely are you to consciously eat less than you want?

1 unlikely 2 slightly unlikely 3 moderately likely 4 very likely

M) Do you go on eating binges though you are not hungry?

1 never 2 rarely 3 sometimes 4 at least once a week

N) On a scale of 0 to 5, what number would you give yourself? (0 means no restraint in eating that is eating whatever you want, whenever you want it, and 5 means total restraint, that is constantly limiting food intake and never 'giving in').

0 eat whatever you want, whenever you want it
1 usually eat whatever you want, whenever you want it
2 often whatever you want, whenever you want it
3 often limit food intake, but often 'giving in'
4 usually limit food intake, rarely 'giving in'
5 constantly limit food intake, never 'giving in'

O) To what extent does the following statement describe your eating behaviour?

'I start dieting in the morning, but because of any number of things that happen during the day, by evening I have given up and eat what I want, promising myself to start dieting again tomorrow.'

1 not like me 2 little like me 3 pretty good description of me 4 describes me perfectly

Appendix 4

Dietary targets Monitor (Lean et al., 2003)

1. **What kind of bread do you usually eat? (Please mark one option)**
   - 1- white
   - 2- brown, granary, wheatmeal
   - 3- wholemeal
   - 4- other kind (please specify)
   - 5- no usual type
   - 6- do not know
   - 7- do not eat bread at all

2. **What do you usually spread on bread? (Please mark an option)**
   - 1- butter
   - 2- hard/block margarine
   - 3- soft margarine
   - 4- reduced fat spread
   - 5- low fat spread
   - 6- no usual type
   - 7- do not spread fat on bread at all

3. **How much do you usually eat in a day? (Please mark an option)**

	Less than 1	1	2-3	4-5	6 or more
- Slices of bread/rolls					
- Biscuits (including chocolate biscuits)					
- Cakes, scones, sweet pies and pastries					

4. **What kind of milk do you usually use for drinks in tea or coffee and on cereals etc? (Please mark an option)**
   - 1- whole milk
   - 2- semi-skimmed
   - 3- skimmed
   - 4- other kind (please specify)
   - 5- no usual type
   - 6- do not drink milk at all

5. **Do you usually take sugar in:**

	YES	NO
(a) Tea		
(b) Coffee		
DO NOT DRINK TEA/COFFEE		

6. **At the table do you: (Please mark an option)**
   - Generally add salt to food without tasting first
   - Taste food and then generally add salt
   - Taste food but occasionally add salt
   - Rarely or never add salt at table

7. **What type of breakfast cereal do you normally eat? (Please mark an option)**
   - High fibre (e.g. All Bran, Branflakes, Shredded Wheat, Muesli, Porridge, Weetabix)
   - Other (e.g. Corn Flakes, Rice Krispies, Special K, Sugar Puffs, Honey Snacks)
   - No usual type
   - Do not eat breakfast cereals at all

	Times: Per day				Per week			Per month		
	6+	4-5	2-3	Once	5-6	2-4	Once	1-3	Less than once	Not at all
Breakfast cereal										
Fresh fruit										
Cooked green vegetables (fresh/frozen)										
Cooked root vegetables (fresh/frozen)										
Salad (raw-vegetables including tomatoes)										
Chips										
Potatoes, pasta, rice										
Oil rich fish (sardines, salmon, mackerel)										
White fish (haddock, cod, whiting)										
Poultry										
Meat products (sausages, ham,)										
Meat (Beef/pork/lamb)										
Cheese										
Beans or pulses										
Sweets, chocolates										
Ice cream										
Crisps, savoury snacks										
Fruit juice (NOT squash)										
Soft-fizzy drinks										
Cakes, scones, sweet pies or pastries										
Biscuits										
Eggs										

9. **In summary:**

(a) How many times do you eat fruit and vegetables or pure fruit juice

[ ][ ] Per day OR [ ][ ] Per week OR [ ][ ] Per month

(b) How many times do you eat oil rich fish

[ ][ ] Per day OR [ ][ ] Per week OR [ ][ ] Per month

(c) How many times do you eat sweets, chocolates, cakes, scones, sweet pies, pastries or biscuits

[ ][ ] Per day OR [ ][ ] Per week OR [ ][ ] Per month

Appendix 5 Food record diary

The following suggestions are helpful in obtaining an accurate record:

- Please note down the day and date, the time, a description and the portion size of the food or drink eaten in every meal or snack

- **Keep going on your usual diet habits** while recording food. Do not modify your food intake patterns during the recording period e.g. number of meals or snacks per day, or the type of foods you usually eat.

- On recording food intake, there are no right or wrong answers…

- After each meal, **record your food intake as soon as possible** to avoid any lapse of memory leading to an inaccurate record.

- Please **record everything eaten or drunk** during the days of the diary. Don't forget to **note down any second helpings**.

- **Remember to include foods or drinks not consumed at home,** for example takeaway/ fast foods, treats/ snacks when visiting others, sweets, crisps and soft drinks.

- **Include as much information as possible** regarding the type of food,
  If homemade, fresh, frozen or tinned.
  The way it was cooked *e.g.* fried (in what type of oil), roasted, boiled, poached, oven baked.
  Whether foods were in batter, breadcrumbs sauce or gravy.
  Brand or shop name

- For portion sizes, some useful descriptions include:
  - Spoon sizes such as teaspoon/tablespoon/dessertspoon/ heaped or level
  - Tin size and amount eaten e.g. half of small tin
  - Number of sweets, biscuits, potatoes, slices of bread
  - If you know the weight of the food in grams or ounces, please note it down

If you have any queries or concerns regarding your food diary, please contact _____ on _____

Thank you for participating in our study.

Day_____ Date_____

Time am/pm	Brand	Description in detail of food include name and quantity	Describe any leftovers

Appendix 6
Pan-European Survey of consumer attitudes to food, nutrition and health
(Kearney and Kearney, 1997)

Now I am going to ask you some questions on a different topic.
DO NOT PROMPT FOOD OR HEALTH

Q1 There are many reasons why we choose the foods that we eat. Looking at this list, which would you say has the greatest influence on your choice of foods? Which has the second greatest influence? Which has the third greatest influence?
SHOW CARD. ROTATE LIST

	Greatest influence	2nd greatest influence	3rd greatest influence
Quality of freshness of food	1	2	3
Habit or routine	1	2	3
Price of food	1	2	3
What my family or spouse or partner will eat	1	2	3
Trying to eat a healthy diet or balanced diet	1	2	3
Taste of food	1	2	3
Convenience in preparation	1	2	3
Presentation or packaging	1	2	3
Slimming	1	2	3
Vegetarian or other special eating habits	1	2	3
Prescribed diet	1	2	3
Content of additives or colours or preservatives	1	2	3
My Cultural or Religious or Ethnic Background	1	2	3
Availability of food	1	2	3
Someone else decides on most of the food I eat	1	2	3
Other PLEASE SPECIFY.....	1	2	3
Do not know	1	2	3

Q2 There is no one definition of healthy eating. Can you tell me please how you personally would describe healthy eating?
RECORD WHAT THE RESPONDENT SAYS.
PROBE "WHAT WOULD HEALTHY EATING INCLUDE OR NOT INCLUDE?
PROBE FOR "ANYTHING ELSE UNTIL RESPONDENT HAS NOTHING ELSE TO SAY.

Q3 Looking at the card, can you tell me from where do you get your information if any, on healthy eating?
SHOW CARD. ROTATE LIST.
PROBE FOR "ANYTHING ELSE" UNTIL RESPONDENT HAS NOTHING ELSE TO SAY.

• Advertising	1
• Information on food packages	2
• Department of Health and other Governmental Bodies	3
• Health professional (such as doctor, nurse, nutritionist, pharmacist)	4
• Leaflets produced by food industry	5
• Women's or family organisations	6
• Books	7
• Articles in Newspapers	8
• Health Food Shop	9
• Programmes or News items on TV and Radio	10
• Magazine articles	11
• School or College or Training	12
• Relatives or Friends or Colleagues	13
• Leaflets in waiting rooms or clinics	14
• Slimming societies	15
• Health Insurance companies	16
• Vegetarian or other food society	17
• Supermarkets	18
• Consumer Organisations	19
• I do not get any information	20
• Others PLEASE SPECIFY	21
• Do not know	22

Q4 There are a number of sources of information on healthy eating, some of which you may trust more than others. Can you tell me please how much you personally would trust or distrust information coming from the following sources, using the card.
SHOW CARD. READ OUT AND ALTERNATE.

	Trust fully	Tend to trust	Tend to distrust	Distrust totally	Do not know
• Department of health and other Government Bodies	1	2	3	4	5
• Advertising	1	2	3	4	5
• Health professional (for example doctor, nurse, nutritionist, pharmacist)	1	2	3	4	5
• Articles in newspapers and magazines	1	2	3	4	5
• Programme on television or radio	1	2	3	4	5
• Information on food packaging	1	2	3	4	5

Q5 Turning to specific foods would you say that people in general should eat MORE, LESS or the SAME AMOUNT than they usually eat of each of the following foods to eat healthier?
READ OUT ALTERNATE

	More	Less	Same	Do not know
• Oily fish (for example, trout or mackerel or salmon)	1	2	3	4
• Savoury Snacks (for example, crisps or peanuts)	1	2	3	4
• Jam or Marmalade or Sugar	1	2	3	4
• Fruit and Vegetables	1	2	3	4
• Bread and Potatoes	1	2	3	4
• Butter or Margarine or Spread	1	2	3	4

Q6 I would like you to tell me to what extent you agree or disagree with each of the following statements, using this card.

SHOW CARD. READ OUT AND ALTERNATE

	Agree Strongly	Tend to agree	Tend to Disagree	Disagree Strongly	Do not know
• I do not need to make any changes to the food I eat, as it is already healthy enough	1	2	3	4	5
• I usually do not think about the nutritional aspects of the types of foods that I eat	1	2	3	4	5
• I frequently look for information on healthy eating	1	2	3	4	5

Q7 Have you ever changed your eating habits to try to eat healthier?

• Yes	1	Q8
• No	2	Q10
• Cannot remember	3	Q10

Q8 Are you eating or trying to eat healthier, these days?

• Yes	1	Q9
• No	2	Q10
• Do not know	3	Q10

Q9 How long have you been eating or trying to eat healthier?

• Less than 6 months	1	Q12
• 6 months or More	2	Q12
• Cannot remember	3	Q12

Q10 In the past month, have you thought about changes you could make to eat healthier?

• Yes	1	Q11
• No	2	Q13
• Do not know	3	Q13

Q11 How confident are you that you will make some changes in order to eat healthier during the next month? PROBE FOR DEGREE OF CONFIDENCE

• Very confident	1	Q12
• Quite confident	2	Q12
• Slightly confident	3	Q12
• Not at all confident	4	Q12
• Do not know	5	Q12

Q12 ONLY ASK THOSE WHO CHANGED THEIR DIETS OR WHO HAVE THOUGHT ABOUT MAKING CHANGES.

Using this card, ca you tell me please, what are those changes?

SHOW CARD. ROTATE LIST. PROBE FOR "ANYTHING ELSE" UNTIL RESPONDENT HAS NOTHING ELSE TO SAY.

• Take less sugar or sugary foods and drinks	1
• Avoid foods with additives or colours or preservatives	2
• Eat less of fatty foods	3
• Drink less alcohol	4
• Eat a smaller amount of food	5
• Eat less processed foods	6
• Avoid missing meals	7
• Eat more fruit or vegetables	8
• Eat more wholemeal or wholegrain foods	9
• Drink more water	10
• Eat a variety of foods	11
• Choose light or low fat or diet or low calorie foods	12
• Eat less meat	13
• Drink less coffee or tea or stimulants	14
• Et more cheese or milk or yogurt	15
• Eat less in between meals	16
• Eat more starch-rich foods	17
• Eat less salt	18
• Eat less bread or potatoes or pasta	19
• Other PLEASE	20
SPECIFY.....	
• None of these	21
• Do not know	22

Q13 Some people believe that healthy eating has specific benefits, some of which are shown on this card. Which, if any, would you personally believe can be achieved by healthy eating?
SHOW CARD. ROTATE LIST. PROBE FOR " ANYTHING ELSE" UNTIL RESPONDENT HAS NOTHING ELSE TO SAY.

Q13b Which one benefit would be most personally significant for you?

Healthy eating can help me to…

	Believe	Most significant
… Control my weight	1	1
… Look attractive	2	2
… Prevent Disease in general (for example heart disease, cancer, etc.)	3	3
… Be fit	4	4
… Live longer	5	5
… Do well at Sport	6	6
… Have plenty of energy	7	7
… Have a better quality of life	8	8
… Stay healthy	9	9
… Other PLEASE SPECIFY……	10	10
… Do not know	11	11
… None of these	12	12

Q14 This card shows some difficulties that people may have in trying to eat healthier. Can you tell me please, which do you think would be the major difficulties for you?
SHOW CARD. ROTATE LIST. PROBE FOR " ANY OTHER MAJOR DIFFICULTIES".

- Irregular working hours 1
- Unappealing food 2
- Cooking skills 3
- Busy lifestyle 4
- Feeling conspicuous among others 5
- Limited choice when I eat out 6
- Taste preferences of family or friends 7
- Too great a change from my current diet 8
- Healthy options not available in shop or canteen or home 9
- Giving up foods I like 10
- Strange or unusual foods 11
- Price of healthy food 12
- Healthy food more awkward to carry home from shops 13
- Healthy food is more perishable 14
- Not knowing enough about healthy eating 15
- Not enough food to satisfy hunger 16
- Lengthy preparation 17
- "Experts" keep changing their minds 18
- Willpower 19
- Storage cooking facilities 20
- Limited cooking facilities 21
- I don't want to change my eating habits 22
- Other PLEASE SPECIFY..... 23
- No difficulty 24

Appendix 7
Physical Activity Questionnaire
1998 Scottish Health Survey (Scottish Office, 1998)

I would like to ask you about some of the things you have done in the past four weeks that involve physical activity, this could be at work (school/college) or in your free time.

1. Thinking about your job in general would you say that you are:

☐ Very physically active
☐ Fairly physically active
☐ Not very physically active
☐ Not at all physically active in your job

2. I would like you to think about the physical activities you have done in the last few weeks (when you were *not* doing your paid job) Have you done any housework in the past four weeks?

☐ Yes
☐ No

3. Have you done any of the following housework: 1 Hoovering; 2 Dusting; 3 Ironing; 4 General tidying; 5 Washing floors and paint work?

☐ Yes
☐ No

4. Some kinds of work are heavier than others. Some examples of heavy housework are: 1 Moving heavy furniture; 2 Spring-cleaning; 3 Walking with heavy shopping (for more than 5 minutes); 4 Cleaning windows; 5 Scrubbing floors with a scrubbing brush. **It does not include everything, these are just examples. Was any of the housework you did in the last four weeks this kind of heavy housework?**

☐ Yes
☐ No

5. During the past four weeks on how many *days* have you done this kind of *heavy* housework? _____ days

6. On the days you did heavy housework, how long did you usually spend?
_____ hours _____minutes

7. Have you done any gardening/do I yourself/ building work in the past four weeks?

☐ Yes
☐ No

8. Have you done any gardening, do I yourself, or building work such as:
1 Hoeing/wedding/pruning; 2 Mowing with a power mower; 3 Planting flowers/seeds; 4 Decorating; 5 Minor household repairs; 6 Car washing/polishing; 7 Car repairs/maintenance**?**

☐ Yes
☐ No

9. Have you done any gardening, do I yourself, or building work such as:
1 Digging/ clearing rough ground; 2 Building in stone/bricklaying; 3 Mowing large areas with a hand mower; 4 Felling trees; 5 chopping wood; 6 Mixing/laying concrete; 7 Moving heavy loads; 8 Refitting a kitchen or bathroom, **or any similar heavy manual work?**

☐ Yes
☐ No

10. During the past four weeks on how many days have you done this kind of heavy manual gardening or DIY (do it yourself)? _____ days

11. On the days you did heavy manual gardening or DIY (do it yourself), how long did you usually spend? ____hours ____minutes

12. I would like you to think about all the walking you have done in the past 4 weeks either locally or away from here. Please include any country walks, walking to and from work and any other walks that you have done. In the past four weeks, have you done a *continuous* walk that lasted *at least* 5 minutes?

☐ Yes
☐ No
☐ Can't walk at all

13. In the past four weeks have you done a *continuous* walk that lasted *at least* 15 minutes?

☐ Yes
☐ No

14. During the past four weeks on how *many days* did you do a walk of at least 15 minutes? _____ days

15. On that day (any of those days) did you do *more than one* walk lasting at least 15 minutes?

☐ Yes more than one walk of 15+ mins (on at least one day)
☐ No, only one walk of 15+ mins a day

16. On how many days in the last four weeks did you do more than one walk that lasted at least 15 minutes? _____ days

17. How long did you usually spend walking each time you did a walk for 15 minutes or more?
_____ hours _____minutes

18. Which of the following best describes your usual walking pace?
☐ A slow pace
☐ A steady average pace
☐ A fairly brisk pace
☐ Or, a fast pace- at least 4 mph
☐ (None of these)

19. Can you tell me if you have done any activities such as: 1 Swimming; 2 Cycling; 3 Workout at gym/Exercise bike/ weight training; 4 Aerobics/ Keep fit/ Gymnastics/ Dance for fitness; 5 Any other type of dancing; 6 Running/Jogging; 7 football/Rugby; 8 Badminton/Tennis; 9 Squash; 10 Exercises (e.g. press ups, sit ups) **during the last 4 weeks,? Include teaching, coaching training and practice sessions.**
☐ Yes
☐ No

20. Which have you done in the last four weeks?
☐ Swimming
☐ Cycling
☐ Workout at gym/Exercise bike/ weight training
☐ Aerobics/Keep fit/Gymnastics/Dance for fitness
☐ Any other type of dancing
☐ Running/Jogging
☐ Football/Rugby
☐ Badminton/Tennis
☐ Squash
☐ Exercises (e.g. press ups, sit ups)
☐ Any others? Which ones? _____

21. Have you done any other sports or exercise not listed above?
☐ Yes Which _____
☐ No

22. Can you tell me on how many days did you do (the activities you have mentioned above) for at least 15 minutes a time during the past four weeks?

(Activity) _____ _____days
(Activity) _____ _____days
(Activity) _____ _____days
(Activity) _____ _____days
(Activity) _____ _____days
(Activity) _____ _____days

23. How much time did you usually spend doing (the activities you have mentioned above) on each day? (Only count times you did it for at least 15 minutes)

_____ hours _____minutes

24. During the past four weeks, was the effort of (the activities you have mentioned above) usually enough to make you out of breath or sweaty?

☐ Yes
☐ No